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Comprehensive analysis of acute flaccid paralysis with and without myelitis in Taiwanese children

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Abstract

Background Acute flaccid paralysis (AFP) is a clinical syndrome marked by the sudden onset of muscle weakness or paralysis, requiring immediate medical intervention due to its potential for significant morbidity and mortality. Despite extensive studies on AFP, comparative analyses between cases with myelitis (M-AFP) and non-myelitis (NM-AFP) remain scarce. This study seeks to address this gap by analyzing demographic, clinical, and etiological distinctions between these groups.

Methods A retrospective study was conducted on 39 pediatric AFP patients diagnosed between 2012 and 2021. Participants were categorized into M-AFP ($n=22$) and NM-AFP ($n=17$) groups based on clinical symptoms and diagnostic imaging. Demographic and clinical characteristics, laboratory findings, and underlying causes were analyzed to identify differences between the groups. Statistical methods were employed to assess significance.

Results Significant clinical differences were observed: limb numbness was more prevalent in M-AFP, while myalgia was more common in NM-AFP. Elevated cerebrospinal fluid white blood cell (CSF WBC) counts were noted in M-AFP cases, though the difference was not statistically significant. Etiologies of M-AFP included multiple sclerosis and enterovirus infections, while NM-AFP involved polymyositis, Guillain-Barré syndrome, and hypokalemic periodic paralysis.

Conclusions This study highlights the distinct clinical and etiological profiles of M-AFP and NM-AFP, emphasizing the need for tailored diagnostic strategies to enhance outcomes in pediatric patients.

Keywords Acute flaccid paralysis, Myelitis, Children

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Introduction

Acute flaccid paralysis (AFP) is a clinical syndrome characterized by the rapid onset of weakness or paralysis with reduced muscle tone in the absence of other obvious causes, such as trauma [1]. It represents a critical medical emergency, particularly in pediatric populations, due to its potential for rapid progression and the risk of significant morbidity and mortality. For pediatricians, timely and accurate identification of the underlying cause is essential, as early diagnosis directly influences treatment outcomes and long-term prognosis [2].

The etiologies of AFP are diverse, ranging from infectious and inflammatory conditions to neuromuscular disorders [3, 4]. Among these, myelitis—an inflammatory condition affecting the spinal cord—is a significant cause of AFP in children, often resulting from viral infections, autoimmune diseases, or demyelinating disorders [5].

However, AFP can also occur without myelitis, with a diverse array of underlying etiologies, including neuromuscular junction disorders, peripheral neuropathies, metabolic disturbances, and even hematologic malignancies. Notable conditions such as Guillain-Barre syndrome (GBS), polymyositis (PM), and hypokalemic periodic paralysis (hypoPP) are critical differential diagnoses that pediatricians must consider promptly [6, 7]. The challenge for clinicians, particularly in pediatric cases, lies in swiftly identifying the correct underlying cause of AFP to initiate the appropriate treatment without delay.

Understanding the demographic and clinical characteristics of pediatric patients with AFP, along with detailed analysis of laboratory findings and identified causes, can significantly enhance diagnostic accuracy and improve patient outcomes. Previous studies have primarily focused on either AFP with myelitis or specific conditions within non-myelitis AFP (NM-AFP). Despite their clinical importance, limited research has compared the two groups directly.

This study aims to address this research gap by analyzing demographic, clinical, and laboratory differences between M-AFP and NM-AFP in pediatric patients. By identifying distinct patterns, this research seeks to enhance diagnostic accuracy and guide timely interventions.

Materials and methods

This study analyzed the demographic and clinical characteristics of patients with acute flaccid paralysis (AFP) with and without myelitis. The primary objectives were to compare the underlying causes, clinical presentations, and diagnostic approaches between AFP patients with myelitis (M-AFP) and those without myelitis (NM-AFP). Data were collected retrospectively from medical records of patients diagnosed between 2012 and 2021 (Fig. 1).

Study design

Participants were categorized into two groups based on the presence or absence of myelitis, confirmed through clinical symptoms and diagnostic imaging. The study followed a retrospective cohort design and adhered to the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines. To ensure comprehensive reporting, we systematically summarized each step of the methodology.

Study population

The study included 39 patients, divided into the M-AFP group ($n = 22$) and NM-AFP group ($n = 17$). Patients with myelitis were identified through MRI findings (e.g., T2-weighted and contrast-enhanced imaging) and characteristic clinical symptoms. Patients without myelitis were diagnosed based on clinical presentations and alternative diagnostic tools.

Data collection

Medical records from January 1, 2012, to December 31, 2021, were screened using a big data system and specific keywords, including:

1. Limb or muscle weakness.
2. Limb or muscle hemiplegia.
3. Immobility.
4. Limb pain.
5. Sensation of "paralysis" or "numbness."

From 14,500 screened records, we identified 39 cases meeting the inclusion criteria. Demographic data (e.g., sex, age of onset) and clinical characteristics (e.g., symptoms at onset, seasonality) were extracted. Laboratory findings, including cerebrospinal fluid (CSF) white blood cell counts and protein levels, were documented. Data were reviewed for accuracy and completeness.

Exclusion criteria: patients were excluded if they had

1. Benign acute childhood myositis (BACM) [8].
2. Muscle or limb pain due to trauma.
3. Temporary limb weakness caused by metabolic or neurological events (e.g., hypoglycemia).
4. Cachexia due to major diseases (e.g., malignancies).
5. "Reluctance to move" caused by psychological factors.

Ethical considerations

The study protocol was approved by the Institutional Review Board of China Medical University Children's Hospital (Approval #DMR-113-041/CMUH113-REC2-029). Written informed consent was obtained from

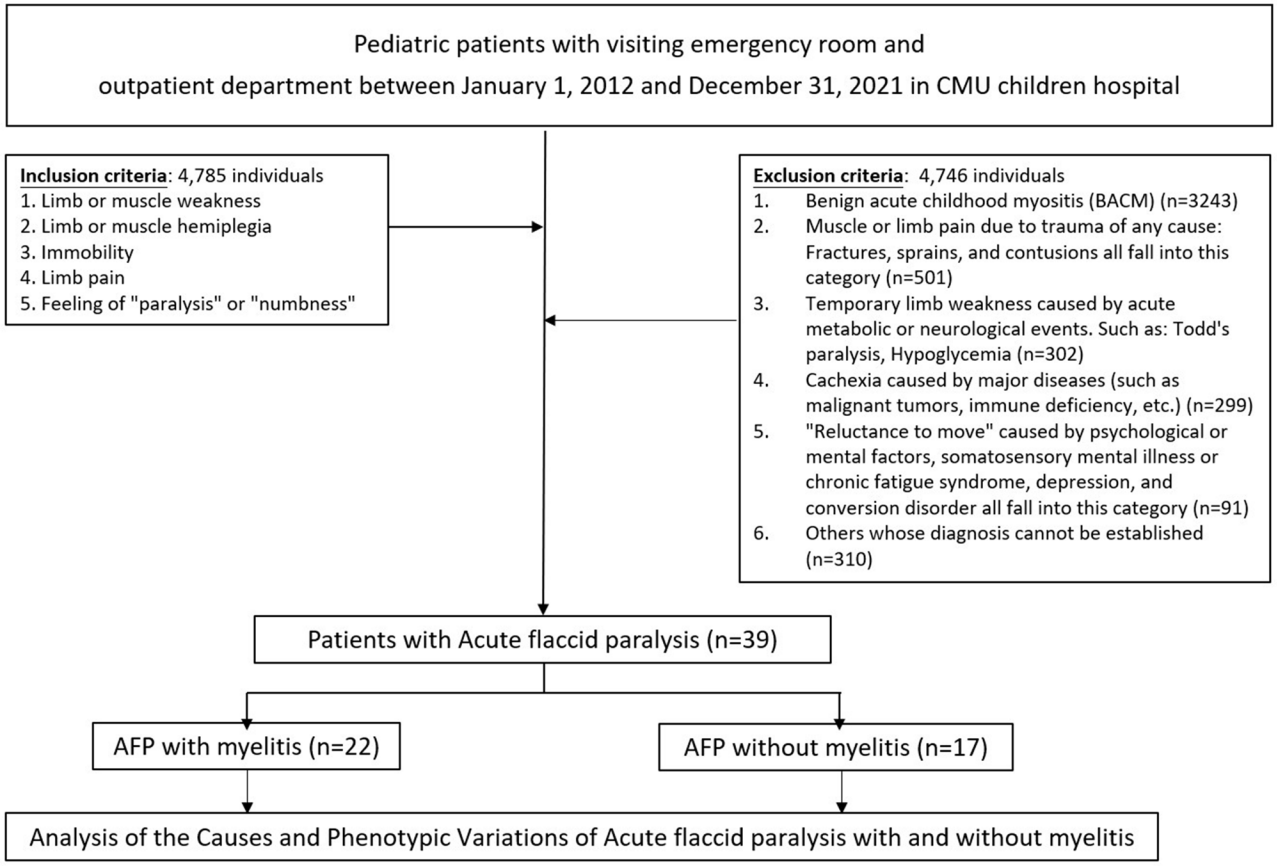

Fig. 1 Flowchart of the study

patients' legal guardians, and all procedures followed the Declaration of Helsinki.

Statistical analysis

All data were validated before analysis to ensure the appropriate application of statistical methods:

Preliminary testing

1. Normality of continuous variables was assessed using the Shapiro-Wilk test.
2. Homogeneity of variance was evaluated with Levene's test.

Analysis approach

1. Continuous variables were analyzed using independent t-tests or Mann-Whitney U tests for non-normal distributions.
2. Categorical variables were compared using chi-square or Fisher's exact tests.
3. A p-value of less than 0.05 was considered statistically significant.

Software

1. Statistical analyses were performed using SPSS Version 25.0 (IBM, Armonk, NY).

Identification of causes: underlying causes were identified using advanced diagnostic tools

M-AFP group

1. MRI findings such as hyperintense lesions on T2-weighted and STIR images.
2. Additional diagnostic criteria included autoimmune markers and viral PCR results.
3. NM-AFP group: neuroimaging, electromyography, and serological tests for conditions like polymyositis and Guillain-Barré syndrome (GBS).

Results

Demographic and clinical characteristics

The demographic and clinical characteristics of the study population are summarized in Table 1. The mean age of onset for M-AFP was 10.5 ± 8.7 years, and for NM-AFP, it was 10.6 ± 4.9 years ($p = 0.96$). There was no significant difference in sex distribution between the two groups ($p = 0.85$). The distribution of onset by season did not

Table 1 Demographic and clinical characteristics of subjects with acute flaccid paralysis with and without myelitis

	Acute flaccid paralysis		*p*-value
	Myelitis (*n* = 22, %)	Non-myelitis (*n* = 17, %)	
Male	11 (50%)	9 (52%)	0.85
Onset age (mean ± SD)	10.5 ± 8.7	10.6 ± 4.9	0.96
Season of Onset			
Spring	3	4	0.42
Summer	8	2	0.08
autumn	3	1	0.42
Winter	8	7	0.75
Illness within 2 months before onset	11	4	0.09
Symptoms at onset			
Limbs Weakness	22	16	0.99
Limbs Numbness	20	5	< 0.001
Myalgia	5	15	< 0.001
Laboratory results			
CSF WBC (mean ± SD)	52.4 ± 62.7	27.6 ± 50.2	0.22
CSF Protein (mean ± SD)	61.8 ± 40.9	68.1 ± 47.6	0.65
Other identified causes of infection	8	0	0.005
Underlying causes identified for diseases	13 (59%)	16 (94%)	0.013
Number of Hospitalization Days (mean ± SD)	20.4 ± 12.6	37.5 ± 34.1	0.71
Diagnosis days (mean ± SD)	29.0 ± 28.5	69.4 ± 49.8	0.81

AFP: Acute Flaccid Paralysis; CSF: Cerebrospinal Fluid; WBC: White Blood Cell; SD: Standard Deviation

show a significant difference between M-AFP and NM-AFP ($p > 0.05$ for all comparisons). All patients with myelitis presented with limb weakness (100%), compared to 16 patients (94%) in NM-AFP ($p = 0.99$). Limb numbness was significantly more common in M-AFP (91% vs. 29%, $p < 0.001$), whereas myalgia was more prevalent in NM-AFP (88% vs. 23%, $p < 0.001$). The mean CSF WBC count was higher in M-AFP (52.4 ± 62.7 cells/μL) compared to NM-AFP (27.6 ± 50.2 cells/μL), but this difference was not statistically significant ($p = 0.22$). The mean CSF protein levels were similar between the two groups (61.8 ± 40.9 mg/dL vs. 68.1 ± 47.6 mg/dL, $p = 0.65$). Additionally, there was no statistically significant difference between the two groups regarding hospitalization days and diagnosis days.

Statistical significance of underlying causes

Tables 2 and 3 present the underlying causes of acute myelitis (M-AFP) and AFP without myelitis (NM-AFP). In M-AFP cases, causes included multiple sclerosis, herpes simplex virus, Behcet's disease, enterovirus, systemic lupus erythematosus, influenza A, spinal subdural hematoma, and Enterovirus D68. In NM-AFP, causes such as polymyositis, porphyria, juvenile dermatomyositis, myasthenia gravis, neuroblastoma, acute lymphoblastic leukemia (ALL), Guillain-Barré syndrome (GBS), spinal cord injury, and hypokalemic periodic paralysis (hypoPP) were identified. Statistical analysis revealed that the prevalence of infections was significantly higher in M-AFP (36% vs. 0%, $p = 0.005$), while NM-AFP had a significantly higher rate of identified underlying causes (94% vs. 59%, $p = 0.013$). This suggests a notable difference in the diagnostic patterns between the two groups.

Discussion

This study provides a comprehensive analysis of the demographic and clinical characteristics, symptoms, laboratory results, and underlying causes of M-AFP and NM-AFP. The findings underscore significant clinical differences between these two groups, offering valuable insights for clinical practice and further research.

Demographic and clinical characteristics

The demographic data revealed no significant difference in the mean age of onset or sex distribution between patients with and without myelitis. Despite no previous research directly comparing demographic data of M-AFP and NM-AFP, studies focused on Guillain-Barré syndrome [9] and AFP of unknown etiology [10] indicate that AFP can affect individuals of any age and gender, highlighting its non-discriminatory nature. Understanding these demographic characteristics is crucial as it broadens the differential diagnosis in clinical settings, ensuring that clinicians consider a wide range of potential underlying conditions when diagnosing AFP.

A notable finding is the significant difference in initial symptoms between M-AFP and NM-AFP. Patients with myelitis frequently presented with limb weakness and numbness, symptoms commonly associated with the inflammation and damage to the spinal cord

Table 2 Identified causes of acute myelitis (M-AFP) for 13 patients, m = myelitis

Patients(Sex/Age)	Causes	Lesion location or plausible cause	Key Diagnostic Tools or Methods/Characteristics
M-AFP1 (F/17y)	M due to MS	T11-L1	spine MRI/ Acute paralysis in legs and Loss of sensation from the abdomen down
M-AFP2 (F/5y)	M due to HSV	T1-L1	spine MRI/ Acute Weakness in legs and Loss of sensation from the chest down
M-AFP3 (F/37y)	M due to Behcet's disease	C5-C8	spine MRI/ Acute Weakness and loss of sensation in arms and legs
M-AFP4 (F/6m)	M due to EV	T9-T12	spine MRI/ Acute Weakness or paralysis in legs and loss of sensation from the abdomen down
M-AFP5 (F/19y)	M due to SLE	T9-T12	spine MRI/ Acute Weakness or paralysis in legs and loss of sensation from the abdomen down
M-AFP6 (M/1y)	M due to Influenza A	T4-T6	spine MRI/ Acute Weakness in legs and Loss of sensation from the chest down
M-AFP7 (M/1y)	M due to spinal subdural hematoma	T11-T12	spine MRI/ Acute paralysis in legs and loss of sensation from the abdomen down, involves bowel and bladder dysfunction
M-AFP8 (M/4y)	M due to EV-D68	C3-C6	spine MRI/ Acute paralysis and Loss of sensation in arms and hands and breathing difficulties
M-AFP9 (F/5y)	M due to EV-D68	T10-L2	spine MRI/ Acute paralysis in legs and Loss of sensation from the abdomen down; Back pain; Bowel and bladder dysfunction
M-AFP10 (M/2y)	M due to EV	C/T/L	spine MRI/ acute quadriparesis
M-AFP11 (M/12y)	M due to EV-71	T11-T12	spine MRI/ Acute Paraparesis in legs
M-AFP12 (F/23y)	M due to MS	C1-C2/T10-T11	spine MRI/ Mild Paraparesis in arm and legs, partly loss of sensation from ankle
M-AFP13 (M/13y)	M due to covid-19	T12-L1	spine MRI/ Acute Paraparesis in legs

MS: Multiple Sclerosis; HSV: Herpes Simplex Virus; EV: Enterovirus; SLE: Systemic Lupus Erythematosus; MRI: Magnetic Resonance Imaging; EV-D68: Enterovirus D68; COVID-19: Coronavirus Disease 2019

Table 3 Identified causes of acute flaccid paralysis without myelitis (NM-AFP) for 16 patients

Patients (Sex/Age)	Causes	Lesion location or plausible cause	Key Diagnostic Tools or Methods/Characteristics
NM-AFP1 (F/16y)	PM	Muscles	MSA panel/ Acute lower limb weakness and pain
NM-AFP2 (F/12y)	PM	Muscles	MSA panel/ Acute lower limb weakness and pain
NM-AFP3 (F/16y)	porphyria	Peripheral Neuropathy	urine PGB (+) and NCV (+)/Acute lower limb weakness and moderate abdominal pain
NM-AFP4 (M/13y)	porphyria	Peripheral Neuropathy	urine PGB (+) /Acute lower limb numbness and serious abdominal pain
NM-AFP5 (M/10y)	JDM	Muscles	MSA panel and The Bohan and Peter criteria/ whole body proximal weakness and pain
NM-AFP6 (M/4y)	MG	neuromuscular junction	general muscle weakness and respiratory muscle weakness
NM-AFP7 (M/7y)	PM	Muscles	MSA panel/ Acute lower limb weakness and pain
NM-AFP8 (F/10y)	Neuroblastoma	Tumor Compression and Spinal Cord Compression	Imaging Studies confirmation and Tissue Biopsy validation/ Acute lower limb weakness and pain
NM-AFP9 (F/3y)	PM	Muscles	MSA panel/ Acute lower limb weakness and pain
NM-AFP10 (F/4y)	MG	neuromuscular junction	AChR-Ab (+) and thymoma detected in chest CT/ptosis
NM-AFP11 (M/17y)	ALL	Bone Pain and Bone Marrow Infiltration caused by ALL	Initial: Complete Blood Count and Peripheral Blood Smear; Confirmed by Bone Marrow Examination and Flow Cytometry/ Acute lower limb weakness and pain
NM-AFP12 (F/16y)	GBS	Peripheral Neuropathy	NCV (+) and CSF examination/ Acute lower limb weakness and numbness, loss of DTR
NM-AFP13 (M/16y)	ALL	Bone Pain and Bone Marrow Infiltration caused by ALL	Initial: Complete Blood Count and Peripheral Blood Smear; Confirmed by Bone Marrow Examination and Flow Cytometry/ Acute lower limb weakness and pain
NM-AFP14 (M/3y)	Spinal cord injury	C7-T1;T5-T6	Whole spine MRI and History Taking /Acute Tetraplegia
NM-AFP15 (M/10y)	hypoPP	Potassium Ion Imbalance resulting in Impaired Muscle Contractility	History Taking and Blood Tests During an Episode/ acute paralysis
NM-AFP16 (M/12y)	hypoPP	Potassium Ion Imbalance resulting in Impaired Muscle Contractility	History Taking and Blood Tests During an Episode/ acute paralysis

PM: Polymyositis; MSA: Myositis-Specific Autoantibodies; PGB: Porphobilinogen; NCV: Nerve Conduction Velocity; JDM: Juvenile Dermatomyositis; MG: Myasthenia Gravis; AChR-Ab: Acetylcholine Receptor Antibodies; CT: Computed Tomography; ALL: Acute Lymphoblastic Leukemia; GBS: Guillain-Barre Syndrome; DTR: Deep Tendon Reflexes; hypoPP: Hypokalemic Periodic Paralysis

characteristic of myelitis [11–13]. This is contrasted by NM-AFP, which reported higher instances of myalgia, characteristic of idiopathic inflammatory myopathies such as polymyositis and juvenile dermatomyositis [14, 15]. This differentiation in symptoms guides clinicians towards the appropriate diagnostic pathway and helps prioritize investigations that can confirm the underlying cause.

Laboratory results

Analysis of CSF showed higher mean WBC counts in M-AFP compared to NM-AFP, although the difference was not statistically significant. Elevated CSF WBC counts in myelitis patients can indicate an inflammatory or infectious process within the CNS [16]. While the study did not find significant differences in CSF protein levels between M-AFP and NM-AFP, elevated protein levels remain a critical marker in diagnosing CNS pathologies [17, 18].

Underlying causes

The etiology of AFP in our cohort was diverse. In M-AFP, conditions such as multiple sclerosis, herpes simplex virus, Behcet's disease, systemic lupus erythematosus, influenza A, spinal subdural hematoma, enterovirus-D68, and enterovirus A71 were identified (Fig. 2) [17, 19–21]. This wide spectrum of infectious, inflammatory, and autoimmune conditions leading to myelitis underscores the need for comprehensive diagnostic approaches, including MRI and specific laboratory tests (e.g., CSF analysis, viral PCR tests, autoimmune markers, and other blood tests) to accurately identify the underlying cause.

Conversely, NM-AFP presented a broader array of underlying causes, highlighting the heterogeneity of this patient group. Conditions such as PM, porphyria, JDM, myasthenia gravis, neuroblastoma, ALL, GBS, spinal cord injury, and hypoPP were identified [22–25]. The variety of these conditions indicates that clinicians must maintain a high index of suspicion for a wide range of potential etiologies, including hematology and oncology diseases like ALL and neuroblastoma [26, 27]. This is crucial because hematologic disorders can present with neurological manifestations that may initially be misinterpreted as primary neurological conditions.

Clinical significance of hematologic disorders: examples of ALL and neuroblastoma

Hematologic disorders, such as neuroblastoma and ALL, are particularly noteworthy due to their potential to cause significant morbidity and mortality if not promptly diagnosed and treated. ALL can present with various neurological manifestations, such as headaches, seizures, and focal neurological deficits, due to leukemic infiltration, treatment-related neurotoxicity, or secondary infections.

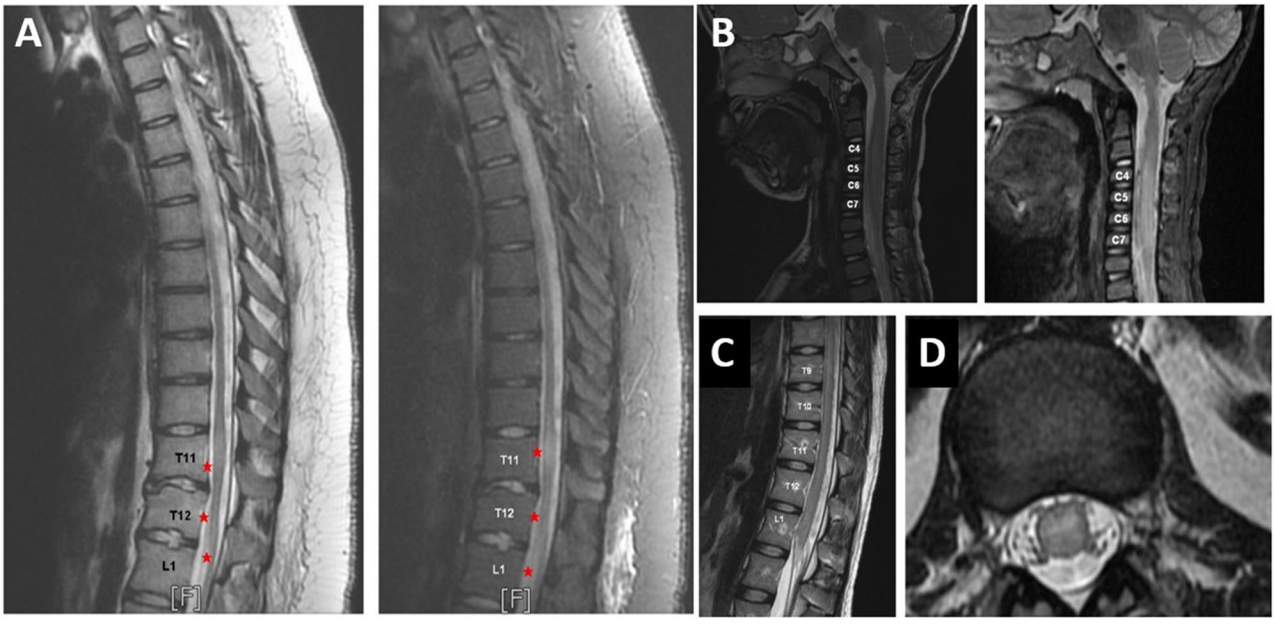

Fig. 2 Detailed Interpretation and Figure Legends for MRI Images of selective patients with Myelitis. (**A**) Sagittal T2W (left) and STIR (right) MRI of the thoracolumbar spine in a 17-year-old female with multiple sclerosis. Vertebral levels T11, T12, and L1 are labeled, with red stars indicating hyperintense lesions due to demyelination. (**B**) Sagittal T2W (left) and STIR (right) MRI of the cervical spine of a 4-year-old male with Enterovirus D68 infection. Vertebral levels C4, C5, C6, and C7 are labeled, showing hyperintense signals indicative of myelitis. (**C**) Sagittal and axial T2-weighted MRI of the thoracolumbar spine of an 18-year-old female with Systemic Lupus Erythematosus. Vertebral levels T9, T10, T11, T12, and L1 are labeled. The sagittal view shows hyperintense lesions, and the axial view at T12-L1 highlights spinal cord inflammation consistent with myelitis. MRI, Magnetic Resonance Imaging; T2W, T2-Weighted; STIR, Short Tau Inversion Recovery; MS, Multiple Sclerosis; EV-D68, Enterovirus D68; SLE: Systemic Lupus Erythematosus

These symptoms can mimic other neurological conditions and lead to an initial misdiagnosis as a primary neurological disorder rather than a hematologic malignancy [28]. Nonetheless, the presentation of AFP in ALL can often be subtle, requiring a high index of suspicion.

Neuroblastoma is a malignant tumor arising from neural crest cells, typically affecting young children. It can lead to AFP through various mechanisms, including direct compression of the spinal cord or nerve roots, paraneoplastic syndromes (e.g., opsoclonus-myoclonus syndrome), and infiltration of the spinal canal (Fig. 3) [29, 30]. This tumor's diverse presentations often include acute weakness or paralysis, sometimes leading to initial misdiagnosis as a primary neurological disorder [30, 31]. By using neuroblastoma and ALL as examples, we underscore the importance of considering hematologic disorders in the differential diagnosis of AFP. This approach ensures that clinicians can identify and manage these potentially life-threatening conditions effectively, improving patient outcomes.

Diagnostic challenges and tools for NM-AFP

Compared to M-AFP, the diagnosis of NM-AFP often takes longer due to several clinical challenges. First, NM-AFP is more etiologically diverse, encompassing inflammatory, metabolic, neuromuscular, and neoplastic conditions. Many of these disorders present with nonspecific or subtle early symptoms that can mimic benign or self-limiting illnesses, making early clinical suspicion difficult. For example, inflammatory myopathies may initially resemble viral myositis, while neuromuscular junction disorders like myasthenia gravis may be mistaken for behavioral fatigue [24]. In addition, hematologic malignancies, such as acute lymphoblastic leukemia (ALL) or neuroblastoma, may present without clear neurological signs initially, delaying definitive diagnosis [26, 27].

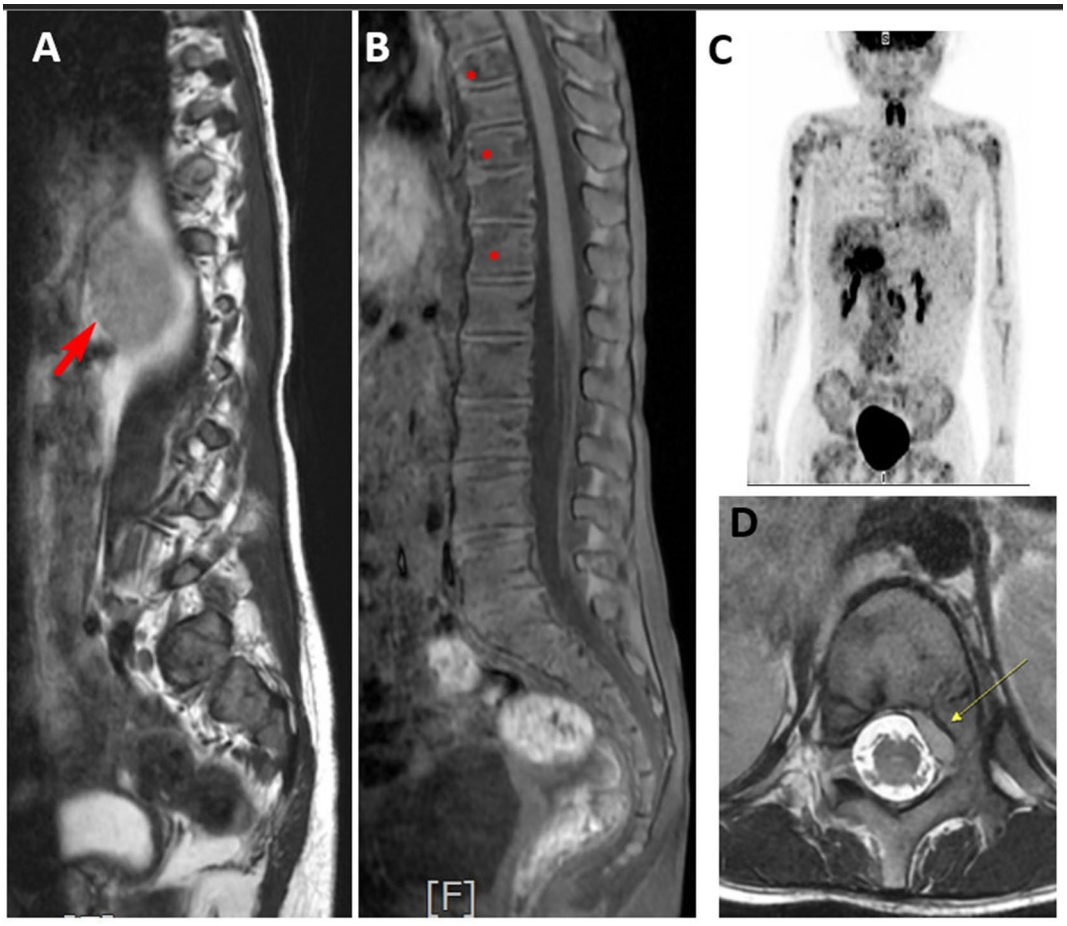

Fig. 3 A 4-year-old girl with bone metastasis from right adrenal neuroblastoma. (**A**) Sagittal MRI showing a large, heterogeneous mass in the right adrenal gland, indicated by the red arrow. (**B**) Sagittal MRI showing multiple vertebral lesions with low itensities, marked by red asterisks (T7, T9 and T11). These lesions show altered signal intensity compared to normal vertebral marrow, indicating metastatic involvement. (**C**) F-18 FDG Whole Body PET Scan: The areas of increased uptake correspond to sites of active tumor growth, including the primary adrenal mass, spine, and possibly other distant organs such as the liver or lymph nodes. The overall pattern of distribution suggests extensive dissemination of neuroblastoma cells. (**D**) Axial MRI at the T12 level showing a metastatic spread of neuroblastoma mass in the left epidural space (yellow arrow), leading to neurological symptoms such as pain, weakness and sensory deficits. MRI, Magnetic Resonance Imaging; F-18 FDG, Fluorodeoxyglucose (F-18); PET, Positron Emission Tomography

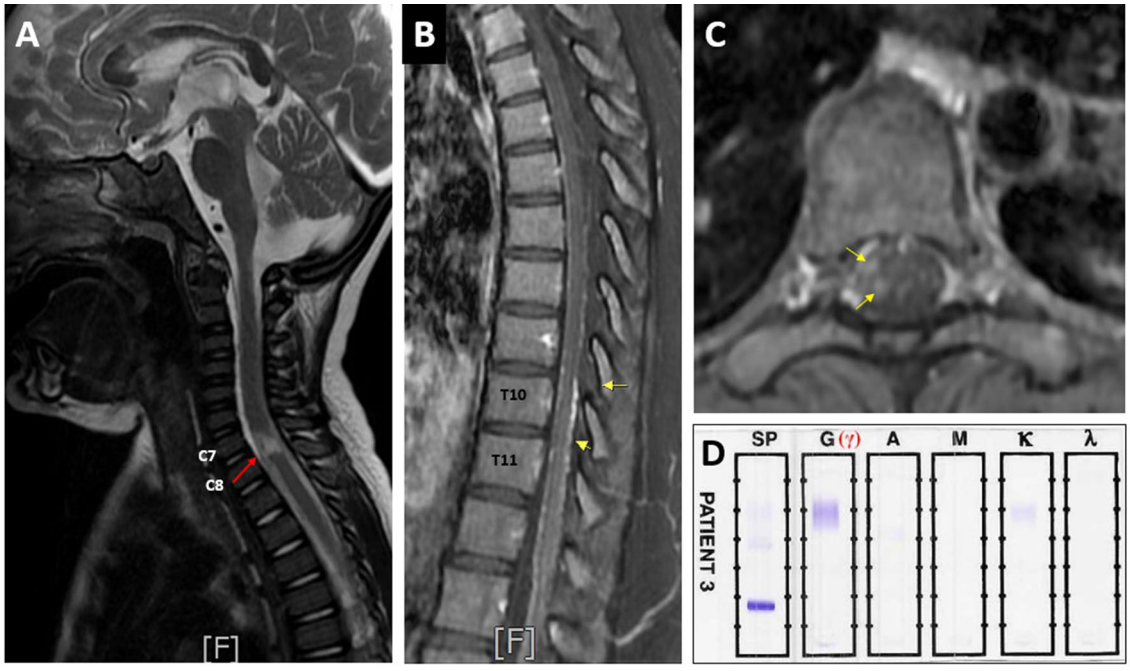

Fig. 4 MRI and Electrophoresis Findings in Patients with Traumatic Injury (**A**) and Guillain-Barre Syndrome (**B-D**): (**A**) The Sagittal T2-weighted MRI of a 3-year-old boy who suffered from a traffic injury. The red arrow indicates a visible injury at the C7-C8 vertebral level, suggesting a spinal cord injury secondary to the accident. (**B**) The Sagittal T2-weighted MRI of a 17-year-old girl with GBS. Yellow arrows highlight spinal nerve root enhancement at the T10 and T11 vertebral levels, indicating inflammation and demyelination associated with GBS. (**C**) Yellow arrows in the Axial T2-weighted MRI indicate nerve root enhancements, consistent with the inflammatory process of GBS. (**D**) The gel of cerebrospinal fluid protein electrophoresis shows an abnormal gamma (γ) band, indicative of an immune response, the autoimmune nature of GBS. MRI, Magnetic Resonance Imaging; GBS, Guillain-Barre Syndrome; T2W, T2-Weighted

Second, early-stage imaging (e.g., spinal MRI) often yields normal or inconclusive findings in NM-AFP, unlike M-AFP where spinal cord lesions are often visible [17, 18]. Consequently, diagnosis often requires advanced and sequential testing, including muscle biopsy, serologic antibody panels, electromyography (EMG), and nerve conduction studies, which are not always immediately accessible in pediatric settings [32, 33].

Third, the initial presentation of conditions such as hypokalemic periodic paralysis or porphyria may mimic post-viral fatigue or functional weakness, causing diagnostic delays [34, 35]. Moreover, clinical overlap between disorders—for example, Guillain-Barré syndrome (GBS) and early juvenile dermatomyositis—can further obscure the diagnosis and delay targeted workups [6, 7, 36].

In our study, we applied a broad range of diagnostic tools to differentiate NM-AFP etiologies. Neuroimaging, such as MRI, was essential for detecting structural abnormalities, including spinal cord injury and tumors (Fig. 4A). Muscle biopsy and serologic tests for muscle-specific autoantibodies were critical in diagnosing polymyositis and juvenile dermatomyositis [32, 33]. For peripheral neuropathies such as GBS and porphyria, EMG, nerve conduction studies, CSF analysis, and immunofixation electrophoresis were essential (Fig. 4B–D) [36, 37]. In suspected hematologic malignancies, initial labs such as a complete blood count and peripheral smear provided important clues, followed by bone marrow biopsy and flow cytometry to confirm diagnoses like ALL [26]. Hypokalemic periodic paralysis was diagnosed through clinical history and serum potassium levels obtained during episodes of weakness [35].

Additionally, emergency settings often lack immediate access to certain specialty tests (e.g., muscle biopsy or autoantibody panels), further delaying diagnostic confirmation [6, 7, 34]. Moreover, emergency physicians may initially attribute weakness to post-infectious fatigue or functional disorders, which can mask the presentation of more serious neuromuscular conditions [6, 7, 37]. Taken together, these findings suggest that the diagnostic process for NM-AFP is inherently more complex and time-consuming. Greater clinical vigilance, comprehensive evaluation protocols, and improved access to diagnostic resources are essential for reducing diagnostic delays and optimizing outcomes in pediatric NM-AFP cases.

Limitations

This study has several limitations. The relatively small sample size may limit the generalizability of the findings. Additionally, the retrospective nature of the study and reliance on medical records may introduce selection bias and inaccuracies in data recording. Furthermore, because

software was used to assist in screening a large number of case data over the past 10 years using keywords instead of manual screening, the number of subjects included and excluded in the experiment is very likely to be different from the actual number of patients. Future prospective studies with larger sample sizes are needed to validate these findings and further explore the differences between AFP with and without myelitis.

Conclusion

This study provides a detailed comparative analysis of AFP with and without myelitis, highlighting significant differences in clinical presentation, laboratory findings, and underlying causes. Patients with myelitis (M-AFP) frequently presented with limb numbness and had higher cerebrospinal fluid white blood cell counts, though the latter was not statistically significant. In contrast, patients without myelitis (NM-AFP) commonly experienced myalgia and had a broader array of underlying conditions, including polymyositis, Guillain-Barre syndrome, and various hematologic disorders. These findings emphasize the importance of a tailored diagnostic approach to enhance the accuracy of diagnosis and the timeliness of interventions. While the study's retrospective nature and small sample size are limitations, the insights gained underscore the need for comprehensive diagnostic strategies to manage AFP effectively. Future research should focus on larger, prospective studies to validate these findings and further refine clinical management protocols, ultimately aiming to improve patient outcomes and reduce the risk of long-term complications.

Abbreviations

AChR-Ab	Acetylcholine Receptor Antibodies
AFP	Acute Flaccid Paralysis
ALL	Acute Lymphoblastic Leukemia
BACM	Benign Acute Childhood Myositis
CNS	Central Nervous System
CSF	Cerebrospinal Fluid
CT	Computed Tomography
DTR	Deep Tendon Reflexes
EV	Enterovirus
EV-D68	Enterovirus D68
F-18 FDG	Fluorodeoxyglucose (F-18)
GBS	Guillain-Barre Syndrome
hypoPP	Hypokalemic Periodic Paralysis
JDM	Juvenile Dermatomyositis
MG	Myasthenia Gravis
MRI	Magnetic Resonance Imaging
MS	Multiple Sclerosis
NM-AFP	Acute Flaccid Paralysis without Myelitis
PCR	Polymerase Chain Reaction
PET	Positron Emission Tomography
PM	Polymyositis
SLE	Systemic Lupus Erythematosus
STIR	Short Tau Inversion Recovery
T2W	T2-Weighted
WBC	White Blood Cell

Supplementary Information

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Supplementary Material 1

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Author contributions

SYH provided treatment to the patient, collected the data and wrote the draft. RHF and YTC participated in the design of the study and wrote the manuscript. ICC and CHL provided their experience for the patient's collection and modified the manuscript accordingly. All authors read and approved the final manuscript.

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Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Consent for discussion of the clinical history was provided by the medical records. The study protocol was approved by the Ethics Review Board of the China Medical University ethics committee (Approval # DMR-113-041/CMUH113-REC2-029). Written informed consent of participation was obtained from the legal guardians. A statement to confirm that all methods were performed in accordance with the ethical standards as laid down in the Declaration of Helsinki and its later amendments or comparable ethical standards.

Consent for publication

The patient's guardians have consented to submission of this study to the journal, and we have obtained a written informed consent.

Competing interests

The authors declare that they have no competing interests.

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