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Case Report
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Chronic Progressive Lower Motor Neuron Tetraparesis in an 11-Year-Old Child: A Rare Case

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Abstract

Background: Chronic progressive tetraparesis with Lower Motor Neuron (LMN) type is a rare neurological condition in children, characterized by progressive weakness of all four limbs with signs of lower motor neurons. Some examples of neuromuscular disorders in children include Guillain-Barré syndrome, multiple sclerosis, myasthenia gravis (MG), muscular dystrophy, and myotonic dystrophy. Early identification and accurate diagnosis are crucial for prognosis and long-term management.

Objective: To report a case of a child with chronic progressive tetraparesis of the LMN type in children who presented with atypical clinical features and required several additional tests to establish the diagnosis.

Case Presentation: Case report of an 11-year-old child who presented with the main complaint of weakness in all four limbs that had lasted for the past 5 months, with slow progression. Physical examination revealed hypotonia, hyporeflexia, and no signs of upper motor neuron involvement. The patient required supportive examinations, including MRI, EMG, and cerebrospinal fluid analysis, to support the diagnosis of chronic progressive LMN motor neuron disorder.

Conclusion: The diagnosis points to progressive chronic LMN disorder. Several relevant differential diagnoses include Duchenne Muscular Dystrophy (DMD), neuropathy caused by anti-TB drugs (isoniazid), Guillain-Barré Syndrome (GBS), Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP), and possibly tuberculous spondylitis. However, the absence of EMG results, spinal MRI, or muscle biopsy means that a definitive diagnosis cannot yet be established.

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INTRODUCTION

Paralysis can be a manifestation of various medical conditions with a wide spectrum of severity, ranging from mild to severe cases. Triggering factors include infection, injury, autoimmune processes, malignancy, and certain metabolic disorders. Paralysis can occur in individuals of all ages and involve partial to complete muscle involvement. Its onset can be sudden, as in Bell's palsy, or gradual, as in stroke. Disorders of the nervous system or muscles, such as Guillain-Barré syndrome, multiple sclerosis, myasthenia gravis, and muscular dystrophy, are often the primary causes. In addition to causes originating from the nervous system, metabolic disorders such as electrolyte imbalance, Addison's disease, and hyperparathyroidism also play a role in triggering paralysis. Clinical complaints may include pain, fever, tingling, and flu-like symptoms, while long-term complications may include muscle wasting (atrophy) and contractures[1].

Clinically, the main signs of paralysis can be muscle weakness, hypotonia, and atrophy due to lower motor neuron (LMN) damage, or spasticity, hypertonia, and hyperreflexia associated with upper motor neuron (UMN) dysfunction [2]. Paralysis in pediatric patients can originate from either the central or peripheral nervous system, so the clinical evaluation process must involve analysis of muscle weakness from both sources [1]. Table 1 explains the difference between LMN and UMN, and it's time-related.

Various neuromuscular diseases, including Guillain-Barré syndrome, multiple sclerosis, myasthenia gravis, muscular dystrophy, and myotonic dystrophy, can trigger paralysis in children. In addition, metabolic factors such as electrolyte disorders, Addison's disease, and hyperparathyroidism also contribute [3].

The following supporting tests should be performed to diagnose tetraparesis in children, as shown in Table 2:

Table 1. Differential Diagnosis of Muscle Paralysis

	Brain	Medula Spinalis	Cornu Anterior	Peripheral Nerves	Muscle Nerve Junction	Muscle
Acute	Gangguan Vaskular	Mielitis Transversa	Poliomielitis	Síndrome Guillain-Barré	Miastenia Gravis (MG)	Miostitis
Episodic	Multiple Sclerosis	Multiple Sclerosis		Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP)	MG	Paralisis Periodica
Subacut	Tumor	Tumor	Atrophy Muscular Spinal (AMS)	CIDP	MG	Dermatomiositis
Chronic	Ensefalopat Metabolik	Tumor	AMS	Neuropati Metabolik	MG	Polimiositis

Source: Braaten, 2018

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Table 2. Supporting Examination for Muscle Paralysis

Deseases	Creatine Kinase (CK)	Nerve Conduction Velocity	Electromi-ography (EMG)	Repetitive Stimulation	Muscle Biopsy
SMA	Normal	Normal	Diagostic	Normal	Neuropaty
Neuropati	Normal	Diagnostic	Neuropaty	Normal	Neuropaty
MG	Normal	Normal	Normal	Diagnostic	Normal
Miopati	Increasing	Normal	Miopaty	Normal	Diagostic

Source: Braaten, 2018

CASE PRESENTATION

An 11-year-old male patient complained of weakness in all extremities that had lasted for five months. Initial symptoms included fever, vomiting, dizziness, joint pain, and dry lips. Initial laboratory tests were within normal limits. Symptomatic treatment resolved the fever, but the patient's weakness progressively worsened and spread to the legs, arms, and neck. The patient had previously been diagnosed with tuberculosis and had undergone treatment with anti-tuberculosis drugs (OAT), but his clinical condition worsened. Currently, the patient is bedridden. The patient is alert and can eat and swallow without choking. The patient complains of headache when the body is lifted, accompanied by generalized cramps throughout the body, including the neck and both arms, which clinically resemble seizures due to excessive muscle contraction. Elimination functions are within normal limits, as evidenced by the ability to urinate without incontinence and to have regular bowel movements. Currently, the patient reports no dizziness or fever.

On examination, the patient was in moderate pain, alert and oriented, with a blood pressure of 100/60 mmHg, pulse rate of 100 beats per minute, respiratory rate of 20 breaths per minute, temperature of 36.7°C, and oxygen saturation of 98% on room air. Weight 28.5 kg, height 145 cm. The patient's nutritional status was normal. Physical examination of the head/neck was normal. Physical examination of the thorax revealed no abnormalities on inspection, palpation, percussion, or auscultation. The entire abdominal examination procedure, including inspection, auscultation, palpation, and percussion, revealed no abnormalities. Neurological examination revealed isocoric pupils measuring 2 mm/2 mm, meningeal irritation signs including neck stiffness and negative Kernig's test, cranial nerve VII within normal limits, cranial nerves IX and X: dysphonia (+), cranial nerve XI: paresis (+) of the sternocleidomastoid and trapezius muscles, and cranial nerve XII within normal limits. Motor function: C5 4/4, C6 4/4, C7 4/4, C8 4/4, T1 4/4, L2 3/3, L3 3/3, L4 3/3, L5 3/3, S1 3/3.

Laboratory tests during treatment revealed elevated SGOT and SGPT levels, and shortened LED. The Mantoux test was negative. CT scan of the head and neck with contrast showed no infarct, hemorrhage, mass, or infectious process in the brain parenchyma, and no fractures in the visualized bones, as shown in figures 1 and 2.

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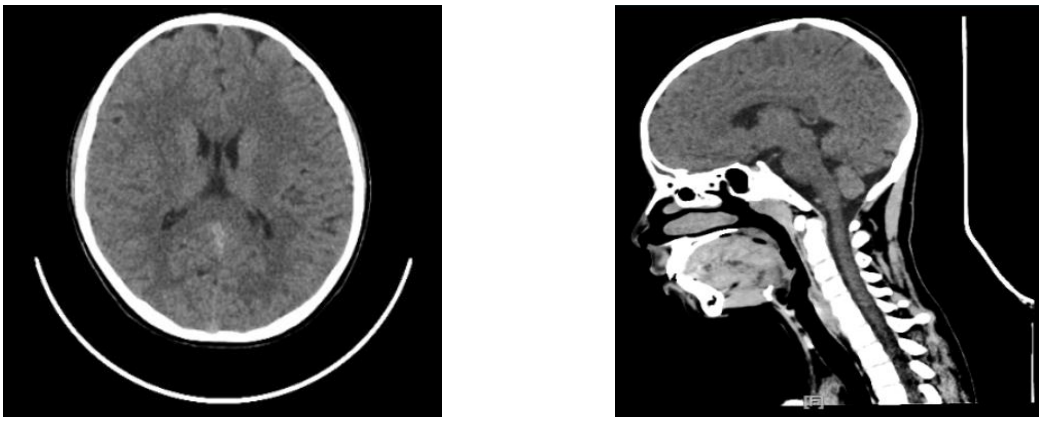

Figure 1. Head and Neck CT Scan Results without Contrast

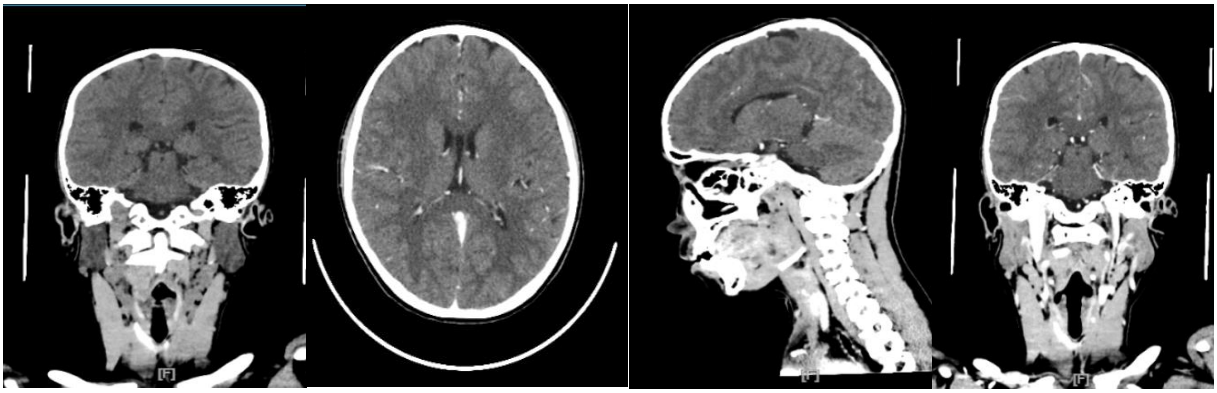

Figure 2. Head and Neck CT Scan Results with Contrast

DISCUSSION

Manifestations of LMN syndrome include muscle weakness and atrophy, accompanied by decreased tendon reflexes, without any sensory system impairment. This condition can be caused by damage to the anterior horn cells of the spinal cord or motor axons and their myelin. Clinical entities in this spectrum include genetic conditions such as Spinal Muscular Atrophy (SMA), Distal Hereditary Motor Neuropathy (dHMN), LMN variants of Motor Neuron Disease (MND), and autoimmune disorders such as Multifocal Motor Neuropathy (MMN) and motoric variants of CIDP [4]. As described on flow chart 1 and its narration, below:

1. Spinal Muscular Atrophy (SMA)

A genetic disorder with an autosomal recessive inheritance pattern characterized by a variety of phenotypes. SMA is a neuromuscular disease characterized by progressive muscle weakness and hypotonia, resulting from degeneration of alpha motor neurons. The age of onset greatly influences early symptoms: in infants and toddlers, severe hypotonia is often accompanied by difficulty swallowing and breastfeeding, while in older children, walking disorders and a tendency to fall frequently are common.

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Muscle weakness and atrophy in SMA are usually symmetrical, more dominant in the lower limbs than the upper limbs, and more prominent in the proximal muscles than the distal muscles. Genetic testing is currently considered the gold standard for diagnosing SMA[5].

2. Distal Hereditary Motor Neuropathy (dHMN)

dHMN is classified as a heterogeneous disease, with differences in clinical presentation and genetic background in each case. Its characteristic features are weakness in the distal muscles of the limbs accompanied by slowly progressive atrophy, with very few or no sensory complaints. Genetic testing, such as a hereditary neuropathy panel or whole-exome sequencing, can detect pathogenic variants in genes associated with dHMN (including HSPB1, HSPB8, GARS1, BSCL2, IGHMBP2, DYNC1H1, TRPV4, and others). These tests form the basis for a definitive diagnosis when clinical findings and electrophysiological results indicate the presence of hereditary motor neuropathy[7].

3. Motor Neuron Disease (MND)

The course of MND varies between individuals and is influenced by the specific subtype of the disease. Initially, MND can cause degeneration of specific motor neuron populations, leading to dysfunction of the corresponding muscle groups. As the disease progresses, degeneration can spread to additional muscle groups, leading to more complex disorders. The availability of next-generation sequencing (NGS) technology has improved diagnostic accuracy and our understanding of genetics, although effective therapies remain limited. These developments are expected to lead to more accurate, specific, and individualized management strategies in the future [2].

4. Multifocal Motor Neuropathy (MMN)

MMN is a slow progressive motor neuropathy characterized by asymmetric muscle weakness without sensory involvement. The typical manifestation of MMN is asymmetric motor weakness without sensory involvement. Diagnosis is based on EFNS/PNS criteria, which include identification of conduction block on NCS examination with normal sensory function, and may be supported by findings of increased protein in CSF analysis[8]. Differential diagnoses, including Guillain-Barré syndrome and hereditary neuropathy, should still be considered[9].

5. Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP)

CIDP in children is a rare autoimmune disease characterized by demyelination of the roots and peripheral nerves, with the main symptoms being symmetrical weakness of proximal and distal muscles, accompanied by loss of deep tendon reflexes and gait disturbance, sometimes preceded by infection. Diagnosis is supported by electrophysiological findings, CSF analysis, and MRI, with most pediatric patients responding well to immunotherapy [10].

This case presents a child with the main complaint of progressive weakness in all limbs (tetraparesis) that has lasted for the past five months. Clinical manifestations include neck muscle weakness, inability to walk, loss of phonation, and involvement of several cranial nerves. This pattern of symptoms is consistent with lower motor neuron (LMN) disorders, which can be triggered by various neurological and neuromuscular conditions[4].

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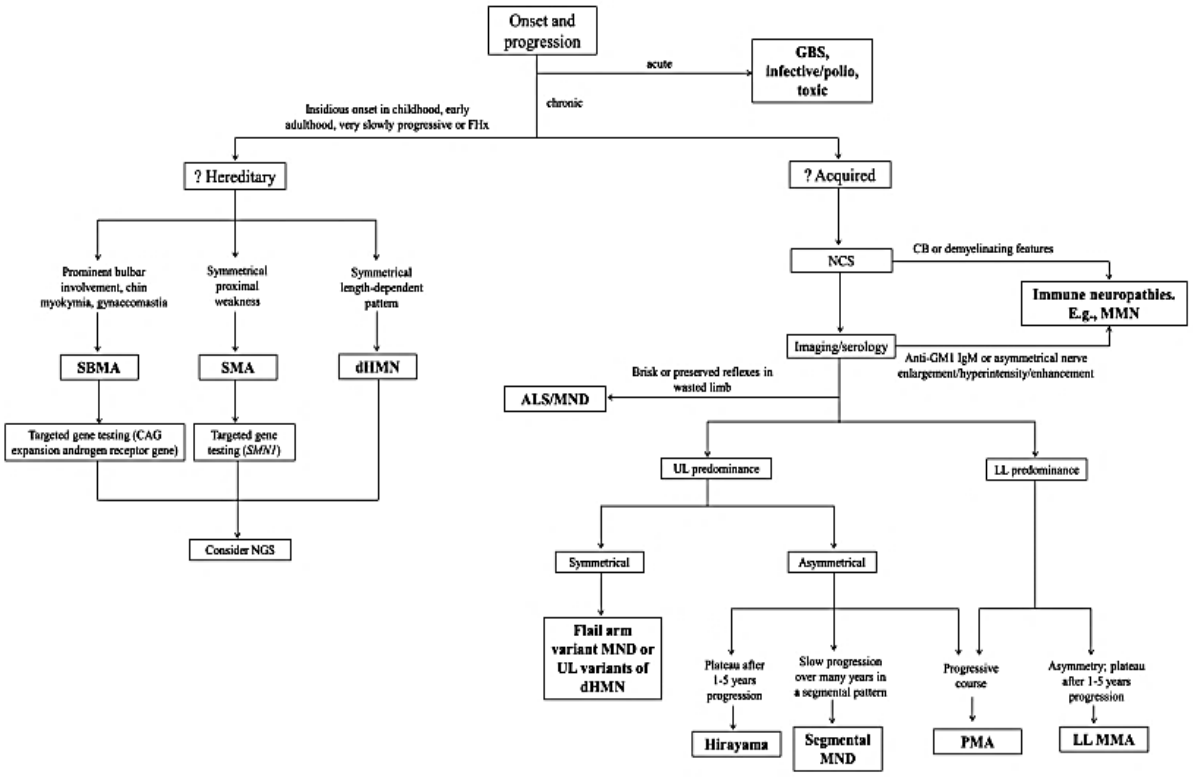

Flow Chart 1. LMN Syndrome Diagnostic Algorithm *Source: Garg, 2017*

One of the main possible diagnoses for this patient is Duchenne Muscular Dystrophy (DMD). DMD is the most common form of muscular dystrophy, characterized by progressive muscle weakness from childhood[11]. This disease is caused by a mutation in the dystrophin gene on the X chromosome, leading to the failure to produce dystrophin protein and resulting in repeated muscle degeneration and replacement by fibrotic tissue and fat. Children with DMD usually show early symptoms such as difficulty getting up from a sitting or squatting position, frequent falls, and a tiptoe gait, before eventually losing the ability to walk at around 10–12 years of age. In advanced stages, the heart and respiratory muscles are also affected, leading to dilated heart failure or respiratory insufficiency as the main causes of mortality[11]. The diagnosis of DMD in children is established through a combination of newborn screening using creatine kinase (CK) testing or genetic testing. Several countries have tried this approach, although ethical considerations, risks of false positives, and healthcare service readiness still need to be taken into account. The examination is followed by confirmation through DMD gene analysis. The gold standard for DMD diagnosis is genetic testing (DMD gene mutation testing), whereas CK testing serves only as an initial marker to trigger further evaluation. Early non-motor symptoms such as speech and developmental delays can also be indicators, so a comprehensive clinical evaluation of motor, cognitive, and social functions is necessary. Additionally, genetic counseling and family screening are important for detecting carriers and providing comprehensive support for families[12].

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Guillain-Barré Syndrome (GBS) can also cause weakness in the extremities, resembling tetraparesis. Unlike this case, which showed months of progression, GBS generally develops rapidly in the acute or subacute phase within days to weeks. In many cases, GBS is preceded by a respiratory or gastrointestinal infection, but this was not observed in this patient [13]. The diagnosis of GBS is primarily established by medical history and clinical examination, with characteristic findings of progressive bilateral weakness and decreased or absent reflexes. Supporting tests such as cerebrospinal fluid analysis (showing increased protein without pleocytosis), electrophysiological studies (showing polyradiculoneuropathy), and neuroimaging or peripheral nerve ultrasonography can help narrow the differential diagnosis and strengthen the suspicion. Although these additional tests are useful, diagnosis should not be delayed if the clinical picture is supportive[14].

A history of anti-tuberculosis drug (ATD) use also raises the possibility of drug-induced neuropathy. Isoniazid, one of the components of first-line ATDs, can induce peripheral neuropathy through a pyridoxine (vitamin B6) deficiency mechanism due to its antagonistic effects on pyridoxine. This risk increases if the patient does not receive vitamin B6 supplementation during therapy[7]. In this patient, there was a history of OAT use that was subsequently discontinued due to worsening weakness, so the possibility of drug-induced neuropathy must still be considered. Neuropathy caused by anti-tuberculosis drugs, particularly isoniazid, is primarily diagnosed clinically by assessing the history of drug use and the appearance of characteristic symptoms of peripheral neuropathy such as tingling, paresthesia, or pain in the extremities; supporting tests such as electrophysiology (EMG/NCS) can help confirm sensorimotor neuropathy, but the diagnosis is primarily established based on the temporal relationship between anti-TB therapy and symptoms, as well as improvement in condition after discontinuation of the drug or administration of vitamin B6 (pyridoxine) supplementation[15].

The patient also had a history of incomplete tuberculosis therapy. This condition raised suspicion of tuberculosis spondylitis as a complication that could potentially compress the spinal cord and cause progressive weakness in the limbs. TB spondylitis is generally characterized by back pain, kyphosis, and neurological deficits due to compression of nerve structures [16]. The diagnosis of tuberculosis spondylitis in children is generally based on a combination of clinical symptoms, radiological examination, and microbiological confirmation. Clinically, patients often present with chronic back pain, stiffness, or postural abnormalities. Imaging techniques such as MRI and CT scans are very helpful because they can detect disc involvement, vertebral body destruction, and paraspinal abscesses earlier than conventional X-rays. The diagnosis can be more firmly established with microbiological or histopathological evidence, such as culture results or molecular testing of infected tissue or fluid. Thus, the diagnosis of TB spondylitis in children emphasizes the integration of clinical, radiological, and specific laboratory findings to confirm the disease and distinguish it from other spinal infections or disorders[17]. However, in this case, the Mantoux test was negative, and the head CT scan did not reveal active lesions, so TB spondylitis could not be confirmed.

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To determine the etiology, supporting tests are essential. Electromyography (EMG) and Nerve Conduction Velocity (NCV) play a role in distinguishing whether the patient's weakness is caused by neuropathic or myopathic disorders[18]. Muscle biopsy can reveal characteristic histopathological findings in muscular dystrophy, such as muscle fiber necrosis, regeneration, and fat infiltration. This examination also serves to rule out non-myopathic causes of muscle weakness[19]. Spinal MRI is necessary to detect potential compressive lesions, such as those that can arise in tuberculous spondylitis or spinal tumors [20]. With a combination of these tests, the differences between DMD, drug-induced neuropathy, and infection can be more clearly determined.

Patient management to date has been symptomatic and supportive, including the administration of neurotropic vitamins (B1, B6, B12), gabapentin for neuropathic pain, physiotherapy to maintain mobility, and high-calorie, high-protein nutritional support (TKTP). This approach is important for maintaining the patient's quality of life while awaiting a definitive diagnosis. If DMD is confirmed, the primary treatment is long-term corticosteroids such as prednisone or deflazacort, which have been shown to slow the loss of walking ability and improve respiratory function[21]. If OAT causes the weakness, therapy is carried out by modifying the drug regimen and supplementing with pyridoxine[22]. Meanwhile, if it is confirmed to be TB-related, treatment of bone or nerve TB infection must be optimized with a complete OAT regimen in accordance with protocol [16].

This case also highlights the importance of a multidisciplinary approach. Patients with suspected DMD or chronic neuromuscular diseases require collaboration between pediatricians, neurologists, medical rehabilitation specialists, physiotherapists, nutritionists, and genetic counselors [21]. This collaboration aims not only to slow the progression of the disease but also to improve the quality of life and support families in long-term planning.

In terms of prognosis, DMD is progressive and fatal, with an average life expectancy into young adulthood. However, the use of corticosteroids and supportive care can prolong survival into the third decade[21]. Conversely, the prognosis for drug-induced or infection-related neuropathy may be better, as muscle weakness may be reversible if the cause is addressed early [13]. Thus, accurate diagnosis in these cases will greatly determine the direction of management and long-term outcomes for patients.

CONCLUSION

This case of chronic progressive tetraparesis of the lower motor neuron (LMN) type in an 11-year-old child is a rare and complex neurological condition that requires a thorough evaluation to determine the correct diagnosis. Clinical symptoms include progressive weakness in all four limbs, accompanied by hypotonia, hyporeflexia, and involvement of several cranial nerves, pointing to chronic progressive LMN disorder. Several relevant differential diagnoses include Duchenne muscular dystrophy (DMD), neuropathy due to anti-TB drugs (isoniazid), Guillain-Barré syndrome (GBS), chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), and possibly tuberculous spondylitis. However, the absence

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of EMG, spinal MRI, or muscle biopsy results prevents a definitive diagnosis at this time.

The current management of the patient is supportive and symptomatic, including the administration of neurotrophic vitamins, gabapentin, and physiotherapy to maintain motor function. In this context, early and accurate diagnosis is crucial, as it guides therapy and influences long-term prognosis. In addition, the involvement of a multidisciplinary team, including pediatricians, neurologists, medical rehabilitation specialists, and genetic counselors, is essential for holistic patient care and family support. Therefore, a systematic and comprehensive follow-up evaluation should be conducted immediately to enable targeted and effective treatment.

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