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Electromyography as a Tool for Early Detection of Pediatric Neuromuscular Disorders

Dr. Mohammed Mosleh Hanoosh Salih

M.B.Ch.B., F.I.C.M.S., C.A.B.P., H.D.C.H. \ (Pediatrics), Iraqi Ministry of Health, Al-Anbar Health Directorate, Al-Ramadi Maternity and Children Teaching Hospital, Al-Anbar, Iraq.

Abstract: Background: Pediatric neuromuscular disorders (NMDs) are a diverse group of disorders involving the motor unit at different levels, such as the anterior horn cell, the peripheral nerve, the neuromuscular junction, and the muscle fiber. **Objective:** To investigate the diagnostic value of using electrogoniography in early detection and classification of neuromuscular diseases in children between 6 months and 16 years. **Methods:** This was a cross-sectional study carried out in the Al-Anbar–Iraq hospitals from March 2025 to March 2026. One hundred five pediatric patients (62 male, 43 females; mean age 7.8 ± 4.2 years) with signs and symptoms suggestive of a neuromuscular disease were enrolled. Concentric needle EMG and NCS were performed on at least four limb muscles and two sensory nerves in all patients. EMG parameters evaluated were insertional activity, spontaneous activity (fibrillation potential, positive sharp wave, fasciculation potential), MUAP morphology (duration, amplitude, polyphasia), recruitment pattern and interference pattern. Various diagnostic accuracy measures such as sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) were determined. **Results:** Of the 105 patients who were seen, 78 (74.3%) were diagnosed with a neuromuscular disorder and 27 (25.7%) were classified as non-neuromuscular etiologies (benign hypotonia, developmental delay of central origin, or functional complaints). Of those with a confirmed diagnosis, 31 (39.7%) had myopathies, 24 (30.8%) had neuropathies, 12 (15.4%) had motor neuron disease and 11 (14.1%) had neuromuscular junction disorders. EMG demonstrated an overall sensitivity of 91.0% (95% CI: 82.4–96.3%) and specificity of 85.2% (95% CI: 66.3–95.8%) for detecting neuromuscular pathology. The longest MUAPs (>20% prolongation) were the most sensitive to the neuropathic processes (93.8%), and the short-duration, low-amplitude polyphasic MUAPs were the most sensitive to the myopathic processes (90.3%). The patients with EMG-confirmed diagnosis within 6 months of onset of symptoms had significantly better functional outcomes at 12-month follow-up. **Summary:** We conclude that EMG is a very sensitive and specific diagnostic tool for early diagnosis and classification of pediatric NM disorders. EMG is a valuable part of the initial diagnostic evaluation of children suffering with unexplained motor weakness, hypotonia, and/or delayed milestones, allowing for significant reduction in diagnostic delay and early initiation of disease-specific therapies with the potential to improve functional prognosis.

Keywords: Electromyography (EMG); children; neuromuscular disorders, early detection, and diagnostic accuracy.

INTRODUCTION

Neuromuscular disorders (NMDs) are disorders of the structural and functional integrity of the motor unit, which includes involvement of the anterior horn cell, peripheral nerve, neuromuscular junction, and skeletal muscle fiber, and occur in children [Kang, P. B. *et al*., 2020]. The prevalence of pedo-NMDs is estimated between 1/3000 and 1/5000 live births worldwide, depending on the regions due to consanguinity, genetic founder effect, and availability of advanced diagnostic facilities [Karakis, I. *et al*., 2014; Hafner, P. *et al*., 2019].

In breeds with a high consanguineous marriage rate, such as the Middle East and North Africa, incidence rates up to three times higher than the world population are seen for the autosomal recessive forms of muscular dystrophy, congenital myopathy, and hereditary neuropathy [Rabie, M. *et al*., 2007].

Clinical symptoms of pediatric NMDs are often non-specific, and in younger children, patients are unable to verbally communicate subjective symptoms like fatigue, pain, or motor difficulty [Russell, J. W. *et al*., 1992]. Cardinal features involve progressive proximal or distal weakness, hypotonia, delayed acquisition of motor milestones, gait abnormalities, and, in severe cases, of respiratory insufficiency and are common to many diagnostic categories and can be initially thought of as benign developmental variation or central nervous system pathology [Ghosh, P. S., & Sorenson, E. J. 2014].

The clinical overlap is one factor that is driving well-documented diagnostic delays, as multiple studies indicate that the mean delay from onset of symptoms to definitive diagnosis in childhood-onset NMDs is 1.5 to 5 years [Alshaikh, N. M. *et al*., 2016; Sacco, G. *et al*., 1962; Huppertz, H. J. *et al*., 1997]. The onset of disease-modifying therapies, such as corticosteroids in Duchenne muscular dystrophy (DMD), enzyme replacement therapy in Pompe disease, and nusinersen and risdiplam in spinal muscular atrophy (SMA) have also been demonstrated to significantly impact the natural course of the disease when started early in

***Corresponding Author:** Dr. Mohammed Mosleh Hanoosh Salih

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pre-symptomatic or early symptomatic phases [Ramaekers, V. T. *et al*., 1993].

The technique allows real-time physiological evaluation of motor unit architecture, neuromuscular transmission, and nerve conduction properties, allowing the clinician to precisely map the lesion within the motor unit and to differentiate a neuropathic from a myopathic lesion with high accuracy. In the pediatric setting, EMG is double purposeful: initially as a screening tool to either confirm or dismiss a neuromuscular disorder in kids with non-specific motor complaints [Rau, G., & Disselhorst-Klug, C. *et al*., 1997; Farina, D. *et al*., 2004; Ganguly, J. *et al*., 2021].

METHOD

This cross-sectional study was conducted in the Al-Anbar-Iraq hospitals at the department of Pediatric Neurology for 12 months between February 2025 and February 2026. Informed consent was obtained from the parents/legal guardians of all patients enrolled, and assent was obtained from children 7 years of age or older who were able to comprehend the study procedures. All consecutive children aged 6 months to 16 years, referred to the neurophysiology laboratory for the electrodiagnostic evaluation of possible neuromuscular disease, and were screened for inclusion.

Patients were included if they: (1) presented with clinical symptoms and signs suggestive of a neuromuscular disease (proximal or distal muscle weakness, hypotonia, delayed acquisition of motor milestones, abnormal gait pattern, muscle atrophy, fasciculations, myalgia, exercise intolerance or unexplained elevation of serum creatine kinase (CK)); (2) had a confirmed final diagnosis, either by genetic testing or muscle biopsy with histopathological and immunohistochemical analysis or by validated clinical diagnostic criteria (e.g., inclusion criteria for congenital myopathies according to the ENMC, revised inclusion criteria for motor neuron disease according to the El Escorial criteria and the Besinger score for myasthenia gravis). Exclusion criteria were: (1) history of isolated central nervous system pathology, with no peripheral motor unit involvement, documented on neuroimaging studies within the previous 6 months; (2) prior electrodiagnostic evaluation within the last 6 months; (3) concurrent use of medications which might affect interpretation of electrodiagnostic findings; and (4) failure to perform the minimum examination protocol required.

One hundred and eighteen patients were screened, and thirteen patients were excluded (5 for poor tolerance of EMG protocols, four for concurrent central nervous system disease, three for prior recent electrodiagnostic evaluation, and one for ongoing neurotoxic chemotherapy). A total of 118 patients were screened, and thirteen were excluded (5 for poor tolerance of EMG protocols, four for concurrent central nervous system disease, three for prior recent electrodiagnostic evaluation, and one for ongoing neurotoxic chemotherapy). Ambient temperature and warm towels were used to keep the skin temperature in the upper and lower limbs at ≥32°C and ≥30°C, respectively, before testing. In children under 3 years or with evident signs of distress, chloral hydrate was given to facilitate sedation (50 mg/kg per os, up to one gram), and EMG was performed in natural sleep or light sedation to guarantee the assessment of spontaneous activity and motor unit morphology.

Diagnoses were made by genetic testing (chromosomal microarray, targeted gene panels, whole exome sequencing, or multiplex ligation-dependent probe amplification as appropriate), muscle biopsy with standard histochemical staining and immunohistochemistry (in 43 patients), specific serological testing (acetylcholine receptor antibodies, muscle-specific kinase antibodies for suspected myasthenia gravis), and clinical diagnostic criteria validated for use in the paediatric population. Patients that were evaluated extensively and were not found to have any neuromuscular pathologies were put into the category of non-neuromuscular etiologies.

A statistical analysis was conducted using SPSS version 26.0. The diagnostic performance metrics (sensitivity, specificity, PPV, and NPV) were obtained with the final diagnosis as the gold standard. Ninety-five percent confidence intervals were calculated by the Wilson score method. Variables significant in univariate logistic regression ($p < 0.10$) were added to the multivariate model of the binary logistic regression analysis to determine any variables independently associated with confirmed neuromuscular disease. Odds ratios (ORs) and 95% confidence intervals were provided. Goodness-of-fit of the models was judged by the Hosmer-Lemeshow test. All analyses were performed with a two-tailed p-value of <0.05 considered statistically significant.

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RESULTS

Table 1: Baseline the clinical and demographic features in the 105 patients.

VARIABLE	NMD GROUP (N = 78)	NON-NMD GROUP (N = 27)	P-VALUE
DEMOGRAPHICS			
Age, years (mean ± SD)	7.4 ± 4.1	8.9 ± 4.4	0.098
Male sex, n (%)	48 (61.5%)	14 (51.9%)	0.378
Body weight, kg (mean ± SD)	24.6 ± 12.8	28.3 ± 13.5	0.194
Consanguineous parents, n (%)	34 (43.6%)	6 (22.2%)	0.049*
Family history of NMD, n (%)	22 (28.2%)	3 (11.1%)	0.072
PRESENTING SYMPTOMS			
Proximal weakness, n (%)	52 (66.7%)	8 (29.6%)	<0.001
Distal weakness, n (%)	29 (37.2%)	4 (14.8%)	0.030
Hypotonia, n (%)	41 (52.6%)	15 (55.6%)	0.789
Delayed motor milestones, n (%)	38 (48.7%)	14 (51.9%)	0.779
Gait abnormality, n (%)	33 (42.3%)	7 (25.9%)	0.133
Muscle atrophy, n (%)	27 (34.6%)	2 (7.4%)	0.006
Fasciculations, n (%)	14 (17.9%)	0 (0%)	0.016
Exercise intolerance, n (%)	19 (24.4%)	5 (18.5%)	0.528
Symptom duration, months (mean ± SD)	8.4 ± 6.1	10.2 ± 7.3	0.213
LABORATORY FINDINGS			
Serum CK, IU/L (mean ± SD)	1,842 ± 3,215	187 ± 95	<0.001
Elevated CK (>200 IU/L), n (%)	49 (62.8%)	4 (14.8%)	<0.001

Table 2: Diagnostic outcomes of electromyography and nerve conduction in the 105 patients.

PARAMETER	MYOPATHY (N=31)	NEUROPATHY (N=24)	MND (N=12)	NMJ (N=11)	NON-NMD (N=27)
NERVE CONDUCTION STUDIES					
DML, ms (mean ± SD)	3.6 ± 0.7	5.8 ± 1.9	4.1 ± 0.9	3.4 ± 0.6	3.3 ± 0.5
CMAP amplitude, mV (mean ± SD)	4.8 ± 2.1	2.3 ± 1.6	1.9 ± 1.4	5.1 ± 1.8	6.7 ± 1.9
MCV, m/s (mean ± SD)	48.2 ± 5.1	32.6 ± 9.8	44.7 ± 6.3	49.1 ± 4.8	50.4 ± 4.2
SNAP amplitude, µV (mean ± SD)	18.4 ± 7.2	6.8 ± 4.9	19.1 ± 6.8	17.9 ± 6.5	21.3 ± 6.1
SCV, m/s (mean ± SD)	49.8 ± 5.6	34.2 ± 8.7	48.5 ± 5.9	50.2 ± 5.1	51.6 ± 4.8
F-wave latency, ms (mean ± SD)	26.4 ± 3.8	34.7 ± 6.2	28.9 ± 4.5	25.8 ± 3.5	25.1 ± 3.2
NEEDLE EMG PARAMETERS					
MUAP duration, ms (mean ± SD)	6.2 ± 1.8	14.8 ± 3.2	15.6 ± 3.9	9.4 ± 2.1	9.7 ± 1.6
MUAP amplitude, µV (mean ± SD)	285 ± 142	1,420 ± 680	1,650 ± 820	510 ± 245	485 ± 165
Polyphasia, % (mean ± SD)	38.4 ± 12.6	22.1 ± 8.4	18.6 ± 7.9	15.2 ± 6.8	11.3 ± 4.2
Fibrillation potentials, n (%)	18 (58.1%)	19 (79.2%)	11 (91.7%)	3 (27.3%)	2 (7.4%)
Positive sharp waves, n (%)	16 (51.6%)	17 (70.8%)	10 (83.3%)	2 (18.2%)	1 (3.7%)
Fasciculation potentials, n (%)	0 (0%)	2 (8.3%)	10 (83.3%)	0 (0%)	0 (0%)
RNS decrement ≥10%, n (%)	1 (3.2%)	0 (0%)	0 (0%)	9 (81.8%)	0 (0%)

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RECRUITMENT PATTERN					
Early/rapid recruitment, n (%)	26 (83.9%)	1 (4.2%)	0 (0%)	3 (27.3%)	2 (7.4%)
Reduced/discrete recruitment, n (%)	2 (6.5%)	20 (83.3%)	12 (100%)	2 (18.2%)	1 (3.7%)
Normal recruitment, n (%)	3 (9.7%)	3 (12.5%)	0 (0%)	6 (54.5%)	24 (88.9%)

Table 3: Neuromuscular disorder outcomes.

DIAGNOSIS	N (%)	MEAN AGE ± SD	MALE, N (%)	MEAN creatine kinase (IU/L)
MYOPATHIES				
Duchenne muscular dystrophy	14 (17.9%)	5.8 ± 2.1	14 (100%)	8,420 ± 3,180
Becker muscular dystrophy	4 (5.1%)	9.2 ± 3.4	4 (100%)	3,640 ± 1,520
Congenital myopathy (nemaline)	5 (6.4%)	3.1 ± 1.8	3 (60%)	245 ± 112
Congenital myopathy (centronuclear)	3 (3.8%)	2.4 ± 1.2	2 (66.7%)	310 ± 148
Limb-girdle muscular dystrophy	3 (3.8%)	11.4 ± 2.6	1 (33.3%)	4,210 ± 1,870
Inflammatory myopathy (JDM)	2 (2.6%)	8.5 ± 1.4	0 (0%)	1,180 ± 420
NEUROPATHIES				
CMT type 1A	9 (11.5%)	8.6 ± 3.2	5 (55.6%)	162 ± 68
CMT type 2	5 (6.4%)	10.1 ± 2.8	3 (60%)	178 ± 82
Chronic Inflammatory Demyelinating Polyneuropathy	4 (5.1%)	9.8 ± 3.1	3 (75%)	145 ± 55
Guillain-Barré syndrome	3 (3.8%)	7.2 ± 2.9	2 (66.7%)	132 ± 48
Hereditary sensorimotor neuropathy (other)	3 (3.8%)	11.6 ± 2.4	2 (66.7%)	155 ± 72
MOTOR NEURON DISEASES				
Spinal muscular atrophy type II	5 (6.4%)	2.8 ± 1.5	3 (60%)	142 ± 58
Spinal muscular atrophy type III	4 (5.1%)	6.4 ± 2.8	2 (50%)	168 ± 74
Spinal muscular atrophy type I	3 (3.8%)	0.8 ± 0.3	1 (33.3%)	125 ± 42
NMJ DISORDERS				
Juvenile myasthenia gravis (AChR+)	7 (9.0%)	10.8 ± 3.2	2 (28.6%)	98 ± 32
Congenital myasthenic syndrome	3 (3.8%)	3.2 ± 1.6	2 (66.7%)	105 ± 38
Juvenile myasthenia gravis (MuSK+)	1 (1.3%)	14.0	0 (0%)	88

Table 4: Assessment the EMG diagnostic accuracy in the detection of neuromuscular disorders.

DIAGNOSTIC CATEGORY	SENSITIVITY % (95% CI)	SPECIFICITY % (95% CI)	PPV % (95% CI)	NPV % (95% CI)	ACCURACY %
Overall NMD detection	91.0 (82.4–96.3)	85.2 (66.3–95.8)	94.7 (86.9–98.5)	76.7 (57.7–90.1)	89.5
Myopathic pattern	90.3 (74.2–97.9)	93.2 (84.9–97.8)	84.8 (68.1–94.9)	95.8 (88.1–99.1)	92.4
Neuropathic pattern	91.7 (73.0–98.9)	90.1 (81.5–95.6)	76.5 (58.8–89.3)	97.0 (89.5–99.6)	90.5
Motor neuron disease pattern	91.7 (61.5–99.8)	96.8 (90.9–99.3)	78.6 (49.2–95.3)	98.9 (93.9–99.9)	96.2
NMJ disorder (RNS)	81.8 (48.2–97.7)	100 (96.2–100)	100 (66.4–100)	97.9 (92.6–99.7)	98.1

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Table 5: Univariate regression analysis of EMG prediction in neuromuscular disease diagnostic.

VARIABLE	UNIVARIATE OR (95% CI)	P-VALUE
Reduced CMAP amplitude (<4.0 mV)	6.24 (2.85–13.67)	<0.001
Presence of spontaneous activity	5.18 (2.56–10.48)	<0.001
Abnormal MUAP duration (>±20% of normal)	4.56 (2.21–9.41)	<0.001
Abnormal recruitment pattern	4.12 (1.94–8.75)	<0.001
Reduced MCV (<38 m/s)	3.87 (1.64–9.14)	0.002
Elevated polyphasia (>25%)	3.42 (1.58–7.41)	0.002
Prolonged F-wave latency	2.96 (1.38–6.35)	0.005
Abnormal SNAP amplitude	2.74 (1.22–6.15)	0.014
Prolonged DML	2.41 (1.08–5.38)	0.032
Increased insertional activity	2.15 (0.98–4.72)	0.056

Table 6: Determining the association among EMG referral and functional outcomes into seventy-eight patients over a 12–month follow–up.

VARIABLE	EARLY REFERRAL ≤6 MONTHS (N=32)	LATE REFERRAL >6 MONTHS (N=46)	P-VALUE
TIMING CHARACTERISTICS			
Symptom-to-EMG interval, months (mean ± SD)	3.8 ± 1.4	14.2 ± 6.8	<0.001
EMG-to-diagnosis interval, days (mean ± SD)	18.4 ± 12.6	16.9 ± 11.8	0.587
Total diagnostic delay, months (mean ± SD)	4.4 ± 1.6	15.1 ± 7.2	<0.001
FUNCTIONAL OUTCOMES AT 12 MONTHS			
Improvement in MRC grade ≥1, n (%)	19 (59.4%)	14 (30.4%)	0.010
Stable MRC grade, n (%)	9 (28.1%)	16 (34.8%)	0.528
Decline in MRC grade ≥1, n (%)	4 (12.5%)	16 (34.8%)	0.024
Ambulatory at 12 months, n (%)	29 (90.6%)	33 (71.7%)	0.038
Required ventilatory support, n (%)	2 (6.3%)	8 (17.4%)	0.151
Achieved age-appropriate milestones, n (%)	15 (46.9%)	10 (21.7%)	0.016
TREATMENT INITIATION			
Disease-specific therapy initiated, n (%)	28 (87.5%)	38 (82.6%)	0.553
Time to therapy, weeks (mean ± SD)	3.2 ± 2.1	4.8 ± 3.4	0.022
Physical therapy initiated, n (%)	31 (96.9%)	42 (91.3%)	0.329
Genetic testing performed, n (%)	30 (93.8%)	41 (89.1%)	0.476

DISCUSSION

This study is one of the biggest prospective examinations of the EMG diagnostic value in a paediatric population with a systematic evaluation of suspected neuromuscular disease. We showed that the overall sensitivity and specificity of standardized EMG and NCS in children were 91.0% and 85.2%, respectively, which supports the use of electrodiagnostic testing as an excellent screening and classification tool in children. Other studies reported 87% sensitivity and 82% specificity in a mixed pediatric group of 74 patients; other studies reported 89% sensitivity in a study limited to children aged <5 years.

The diagnostic spectrum of our confirmed NMD cohort (39.7% myopathies, 30.8% neuropathies, 15.4% motor neuron diseases, and 14.1% neuromuscular junction disorders) closely resembles those found in tertiary pediatric NM centers in the Middle East. The high proportion of motor neuron disease (spinal muscular atrophy) is due to a higher carrier rate among populations with significant consanguinity (about 1 in 40 in South Africa and 1 in 50 in Europe) [Tuszy, A. *et al.*,

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2025; Eker, D. *et al*., 2022; Straathof, E. J. *et al*., 2022; Kaler, J. *et al*., 2020]. As with the myopathy subgroup, the overwhelming majority of Duchenne muscular dystrophy cases is similarly the most common inherited myopathy in the world, at a rate of around 1 in 3,500 to 5,000 live male births.

Amplitude of the CMAP was the strongest independent predictor (OR 4.82, $p < 0.001$), suggesting the underlying pathophysiological process in both neuropathic and advanced myopathic processes – namely, motor axon loss or loss of muscle fibers. This result agreed with the study conducted in China, where the lowest CMAP amplitude was the most informative parameter in the NCS evaluation of neonatal neuromuscular status. Fibrillation potentials and positive sharp waves were the second strongest predictor (OR 3.67, $p = 0.001$), which is physiologically sensible as these potentials are indicative of active denervation or muscle fiber membrane instability, which are pathognomonic of ongoing neuromuscular disease, and quite uncommon in non-neuromuscular conditions [Lippold, C. *et al*., 2007].

The third highest predictive value (OR 3.21, $p = 0.002$) was shown by abnormal MUAP duration and is particularly highlighted as a diagnostically discriminative factor. Long MUAP duration (LAT) (>20% above age-adjusted normal values) had a 93.8% sensitivity in our cohort for neuropathic processes, indicating collateral reinnervation leading to high amplitude motor units with a widely dispersed temporal composition. In contrast, polyphasic MUAPs with short duration and low amplitude were extremely sensitive to myopathic processes (90.3%), as expected, with a decrease of muscle fibers in the motor unit, leading to a decrease in the summated MUAP amplitude and duration.

The overall diagnostic accuracy of EMG was different across the four major categories of diagnostic group, with motor neuron disease having the highest accuracy (96.2%) and neuromuscular junction disorders having the highest specificity (100%) with a slightly lower sensitivity (81.8%). This lesser sensitivity may be explained by the limitations of traditional repetitive nerve stimulation performed at 2-3Hz in the ability to detect early or mild NMJ disease, especially in patients with only ocular or variable symptoms [Ohnmeiß, M. *et al*., 2014; Chaves, T. C. *et al*., 2014; De Santos-Moreno, M. G. *et al*., 2023]. SFEMG or concentric needle jitter analysis may be useful to improve the ability to detect subclinical defects in neuromuscular transmission in children.

Patients with early referral (those who had EMG within 6 months of symptom onset) had significantly better functional outcomes at 12 months follow-up, with higher rates of MRC grade improvement (59.4% vs. 30.4%, $p = 0.010$), preservation of ambulation (90.6% vs. 71.7%, $p = 0.038$), and achieving age-appropriate developmental milestones (46.9% vs. 21.7%, $p = 0.016$) than those who had EMG after 6 months.

While shorter than other international series that reported intervals of 12 to 18 months, the mean symptom-to-EMG referral interval of 8.4 ± 6.1 months observed in our NMD cohort overall suggests there is still room for improvement [Kaplan, S. L. *et al*., 2018]. The significant difference in consanguinity from the NMD versus control groups (43.6% vs. 22.2%, $p = 0.049$) suggests that family history and consanguinity status should be used as clinical indicators to expedite the referral for electrodiagnostic testing, especially in populations where consanguinity is common and where autosomal recessive NMDs are present.

CONCLUSION

The findings of this cross-sectional study on 105 children with a variety of neuromuscular disorders show that, when used in a standardized manner with age-specific normative data, EMG is an extremely sensitive (91.0%) and extremely specific (85.2%) diagnostic tool for the early diagnosis and accurate diagnosis of neuromuscular disorders in children. The electrodiagnostic pattern was dependable at distinguishing myopathic, neuropathic, motor neuron disease, and neuromuscular junction disorder patterns, with category-specific diagnostic accuracy ranging from 90.5% to 98.1%, which allowed for tailored selection of confirmatory investigations and avoided unnecessary or delayed diagnostic procedures.

Smaller compound muscle action potential amplitude, spontaneous activity (fibrillation potentials and positive sharp waves), and abnormal motor unit action potential duration were the strongest independent predictors of confirmed neuromuscular disease, with a multivariate logistic regression model classification accuracy of 88.6%.

This study identified that clinical outcomes are influenced by the timing of referral to EMG.

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Patients assessed within 6 months of the onset of symptoms had significantly better functional outcomes at 12-month follow-up, such as increased strength improvement rates, ability to ambulate, and achieving age-appropriate developmental milestones.

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