

A Study To Correlate Serum Creatine Kinase Level With Motor Function In Children With Duchenne Muscular Dystrophy

Dr. Sheereen Mirza¹ and Dr. Rupesh Masand², Dr. Priyanshu Mathur³

¹PG Resident.

²Professor & Head Department of Paediatrics M.G. Medical College & Hospital, Jaipur.

³Associate Professor Rare Disease and Metabolic Genetics Specialist Department of Pediatrics, SMS Medical College, Jaipur.

ABSTRACT

Background: Duchenne muscular dystrophy (DMD) is a severe X-linked recessive neuromuscular disorder of childhood characterized by progressive proximal muscle weakness and markedly elevated serum creatine kinase (CK) levels, but the relationship between CK and motor function across different severity levels remains uncertain. The objective of this study was to correlate serum creatine kinase levels with motor function in children with Duchenne muscular dystrophy using Manual Muscle Testing (MMT) grades and functional scales.

Methodology: This hospital-based observational and analytical study was conducted in the Department of Paediatrics, Mahatma Gandhi Medical College and Hospital, Jaipur, and included 50 genetically confirmed DMD patients aged 3–18 years. Demographic data, clinical features, MMT grades of neck flexors, shoulder abductors and knee extensors, Brooke and Vignos functional scales and serum CK levels were recorded and analyzed using descriptive statistics, Kruskal-Wallis test with Bonferroni-adjusted Mann-Whitney U test and Spearman and Pearson correlation coefficients.

Results: The majority of participants belonged to the 5–10 years age group (54%), followed by 11–15 years (38%), indicating most commonly observed in middle childhood. The mean age was 9.68 ± 3.285 years with a median of 9 years. Children with lower average MMT scores (<2.0) had markedly elevated CK levels (mean $36,314.31 \pm 26,157.72$ U/L; median 37,832 U/L), whereas those with moderate (2.0–3.0) and higher (>3.0) MMT scores showed progressively lower CK values (means $9,459.82 \pm 3,923.94$ U/L and $5,030.06 \pm 4,693.34$ U/L, respectively), and these differences were statistically significant (Kruskal-Wallis $H = 17.299$, $p = 0.0001$, with all Bonferroni-adjusted pairwise comparisons $p < 0.05$). Serum CK demonstrated a strong, highly significant negative correlation with average MMT score (Spearman $\rho = -0.633$, $p < 0.001$), and similar inverse correlations were observed for neck flexors, shoulder abductors and knee extensors individually, confirming that higher CK levels consistently corresponded to poorer motor function across the examined muscle groups.

Conclusion: Higher serum creatine kinase levels are associated with poorer motor function in children with Duchenne muscular dystrophy, indicating that CK can serve as a useful biochemical marker that complements clinical motor assessments in evaluating disease severity.

Keywords: Duchenne muscular dystrophy; serum creatine kinase; motor function; Manual Muscle Testing; children.

INTRODUCTION

Duchenne muscular dystrophy (DMD) is a severe X linked recessive neuromuscular disorder of childhood characterized by progressive muscle weakness and early loss of independent ambulation.¹ It is caused by dystrophin gene mutations leading to absence or marked reduction of dystrophin, resulting in muscle membrane instability and continuous muscle fiber damage.^{1,2} Clinically, children present with delayed motor milestones, difficulty in running and climbing stairs, frequent falls and Gowers' sign. As the disease advances, complications such as contractures, scoliosis, respiratory involvement and cardiomyopathy develop, making it a life limiting condition.² Regular monitoring of motor function is therefore essential for clinical management.³ Serum creatine kinase (CK) is a muscle enzyme released into the bloodstream following muscle fiber damage.⁴ In DMD, CK levels are markedly elevated due to ongoing muscle breakdown and are widely used as an inexpensive and early diagnostic marker.⁵ However, CK reflects muscle injury rather than actual muscle strength or functional ability. Its levels may fluctuate with physical activity, minor trauma and disease stage, and in later stages of DMD, CK may decline despite worsening weakness due to loss of muscle mass and replacement with fibrofatty tissue.⁶

Motor function in children with DMD is assessed using standardized clinical scales and timed functional tests that evaluate activities such as standing, walking, running and stair climbing. These tools provide objective and reproducible measures of disease severity and are important for monitoring progression, planning physiotherapy and assessing response to emerging therapies.^{7,8} Since disease progression is variable and non-linear, repeated assessments are necessary to capture individual functional decline over time. Although CK indicates ongoing muscle damage and motor scales reflect functional ability, the relationship between them is not clearly established. In early disease, very high CK levels may coexist with relatively preserved motor

function, whereas in later stages CK may fall even as disability worsens. This inconsistency creates challenges in clinical interpretation and highlights the need to understand whether CK can meaningfully reflect functional status.^{9,10} This study aims to investigate the correlation between serum CK levels and motor function in children with DMD to better understand the clinical relevance of CK beyond diagnosis.

METHODOLOGY

This was a hospital-based observational and analytical study conducted in the Department of Paediatrics, Mahatma Gandhi Medical College & Hospital, Jaipur. The study period extended from April 2024 to September 2025 and included all eligible children with Duchenne muscular dystrophy presenting to the department during this interval. All children diagnosed with Duchenne muscular dystrophy were considered for inclusion, provided they met the eligibility criteria and consented to participate. The diagnosis of DMD was confirmed using Multiplex Ligation-dependent Probe Amplification (MLPA) testing for dystrophin gene mutations. The minimum required sample size was calculated as 47 using Fisher's z-transformation for correlation, assuming a two-sided type I error of 0.05, 80% power and an expected correlation coefficient of 0.40 between serum CK and motor function, and the final sample was rounded up to 50 children for feasibility. Inclusion criteria were: (1) genetically confirmed DMD, and (2) written informed consent from parents or legal guardians. Exclusion criteria were: (1) coexistence of major illnesses that could independently affect CK levels, such as liver disease, myositis or acute viral infections.

Data collection and clinical assessment

Consecutive eligible patients were enrolled until the target sample size was reached. For each child, a structured case record proforma was completed, documenting demographic data (age), relevant medical and family history, socioeconomic status and detailed clinical examination findings. Classical DMD clinical features such as Gower's sign, waddling gait, toe walking, difficulty in supine-to-sitting transition and calf hypertrophy were specifically recorded and later analyzed according to MMT categories. Motor function was assessed using Manual Muscle Testing (MMT) based on the Medical Research Council (MRC) grading scale, which grades strength from 0 (no contraction) to 5 (normal strength) with intermediate grades reflecting increasing strength against gravity and resistance.⁷⁵⁻⁷⁷ In this study, MMT was performed for neck flexors, shoulder abductors and knee extensors, and an average MMT score was derived for each patient. In addition, functional status of upper and lower limbs was evaluated using the Brooke Upper Extremity Functional Scale and the Vignos Lower Extremity Functional Scale, respectively, which are commonly applied functional measures in DMD.

Laboratory assessment

Serum creatine kinase levels were estimated in all enrolled patients as part of the standard diagnostic and assessment protocol. CK values were recorded in units per liter (U/L) and were later grouped according to average MMT categories defined as low (<2.0), moderate (2.0–3.0) and high (>3.0) motor function.

Statistical analysis

All collected data were compiled, coded and entered into Microsoft Excel and analyzed using SPSS version 28.0. Descriptive statistics and frequency analysis were used to summarize demographic, clinical, laboratory and motor function variables, with categorical data presented as frequencies and percentages and continuous variables expressed using appropriate measures of central tendency and dispersion. The Chi-square test was used to assess associations between categorical variables. Differences in serum creatine kinase levels across motor function categories were analyzed using the Kruskal-Wallis H test, with post-hoc pairwise comparisons performed using the Mann-Whitney U test with Bonferroni correction. The association between serum creatine kinase levels and motor function scores was assessed using Spearman's rank correlation, and Pearson's correlation was additionally applied after appropriate data transformation. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 50 children with genetically confirmed Duchenne muscular dystrophy were included. The age distribution of children included in the study.

Table 1: Age distribution

Age Group	No.	Percentage
<5	3	6%

5-10	27	54%
11-15	19	38%
>15	1	2%
Total	50	100%
Mean±SD	9.68±3.285	
Median (IQR)	9.00 (7.25 - 11.75)	

The majority of participants belonged to the 5-10 years age group (54%), followed by 11-15 years (38%), indicating that Duchenne Muscular Dystrophy (DMD) cases were most commonly observed in middle childhood. A smaller proportion was seen in children below 5 years (6%) and very few cases were above 15 years (2%), reflecting the progressive nature of the disease and earlier diagnosis. The mean age was 9.68 ± 3.285 years with a median of 9 years, suggesting that most patients were clustered around late childhood.

Table 2: Measurement of Global Development Quotient (%) according to MMT category

MMT category	N	Mean	SD	F-value	p-value
Low (<2)	16	42.94	19.523	18.791	0.001
Moderate (2.0-3.0)	17	57.59	9.193		
High (>3.00)	17	71.82	9.787		
Total	50	57.74	17.774		

The relationship between Global Development Quotient (GDQ) and Manual Muscle Testing (MMT) categories. It was observed that children with low MMT scores (<2) had significantly lower GDQ (mean: 42.94 ± 19.523), while those with moderate MMT (2-3) showed improved GDQ (mean: 57.59 ± 9.193). The highest GDQ values were seen in children with high MMT scores (>3) (mean: 71.82 ± 9.787). The difference across groups was statistically highly significant ($F = 18.791$, $p = 0.001$). This indicates a strong positive association between muscle strength and developmental status. In other words, better motor function is associated with higher developmental outcomes in children with DMD.

Table 3: Presence of Clinical Features according to MMT category

Clinical Features	Low (<2)	Moderate (2.0-3.0)	High (>3.00)	Total	χ^2	p-value
Gower's sign	16	16	7	39	20.520	0.000*
	100.0%	94.1%	41.2%	78.0%		
Waddling gait	12	13	3	28	15.384	0.000*
	75.0%	76.5%	17.6%	56.0%		
Toe walking	15	9	10	34	7.305	0.026*
	93.8%	52.9%	58.8%	68.0%		
Supine to sitting	13	8	3	24	13.368	0.001*
	81.3%	47.1%	17.6%	48.0%		
Hypertrophied muscles	11	11	14	36	1.436	0.488
	68.8%	64.7%	82.4%	72.0%		

* Significant ($p < 0.05$)

The distribution of various clinical features across different MMT categories. It was observed that classical features of Duchenne Muscular Dystrophy (DMD), such as Gower's sign, waddling gait, toe walking and difficulty in supine-to-sitting were significantly more common in children with lower MMT scores indicating poorer muscle strength. Gower's sign was present in 100% of patients in the low MMT group, compared to 41.2% in the high MMT group, showing a highly significant association ($p < 0.001$). Similarly, waddling gait and toe walking were also more prevalent in low and moderate MMT groups with statistically significant differences ($p < 0.05$). Difficulty in supine-to-sitting transition also showed a strong association with lower muscle strength ($p = 0.001$). However, muscle hypertrophy did not show a statistically significant association ($p = 0.488$), suggesting that it may be present across all stages of the disease irrespective of severity. Overall, the table indicates that most clinical features worsen as muscle strength declines and are significantly associated with disease severity.

Table 4: Motor function using MMT Grading

MMT Grade	Neck Flexors		Shoulder Abductors		Knee Extensors	
	No.	%	No.	%	No.	%
0	5	10%	6	12%	3	6%
1	10	20%	7	14%	11	22%
2	8	16%	10	20%	14	28%
3	10	20%	13	26%	13	26%
4	11	22%	10	20%	8	16%
5	6	12%	4	8%	1	2%
Total	50	100%	50	100%	50	100%
Mean±SD	2.60 ± 1.565		2.52 ± 1.474		2.30 ± 1.216	
Median (IQR)	3.00 (1.00-4.00)		3.00 (1.25-4.00)		2.00 (1.00-3.00)	

The distribution of motor function based on MMT grading across three muscle groups: neck flexors, shoulder abductors and knee extensors. The results show that most children had muscle strength clustered around grade 2 to grade 4, indicating moderate muscle weakness. For neck flexors, the majority of patients were in grade 3 (20%) and grade 4 (22%) with a mean score of 2.60 ± 1.565 . Similarly, shoulder abductors showed a concentration in grade 3 (26%) with a mean of 2.52 ± 1.474 . Knee extensors demonstrated comparatively lower strength with the highest proportion in grade 2 (28%) and grade 3 (26%) and a lower mean of 2.30 ± 1.216 , suggesting earlier or more severe involvement of lower limb muscles. The median values further support moderate muscle weakness across all groups. Overall, the findings indicate that proximal muscle groups, especially in the lower limbs are more severely affected, which is consistent with the known progression pattern of Duchenne Muscular Dystrophy.

Table 5: Serum creatine kinase (U/L) levels by average MMT category

Average MMT category	n	CPK mean ± SD	CPK median (IQR)	CPK min-max
Low (<2.0)	16	36314.31 ± 26157.72	37832 (12543-56882)	1070-79456
Moderate (2.0-3.0)	17	9459.82 ± 3923.94	8787 (7234-12765)	2800-18343
High (>3.0)	17	5030.06 ± 4693.34	3456 (2662-4234)	1945-21100

Kruskal-Wallis H = 17.2990

p= 0.0001

The distribution of serum Creatine Phosphokinase (CPK: Creatine Phosphokinase) levels across different MMT (Manual Muscle Testing) categories. It was observed that children with low MMT scores (<2.0) had markedly elevated CPK levels (mean: 36314.31 ± 26157.72 U/L), whereas those with moderate MMT (2.0-3.0) had lower levels (mean: 9459.82 ± 3923.94 U/L) and the high MMT group (>3.0) had the lowest CPK levels (mean: 5030.06 ± 4693.34 U/L). This indicates a clear inverse relationship between muscle strength and CPK levels. The difference across groups was found to be highly statistically significant (Kruskal-Wallis H = 17.299, p = 0.0001). These findings suggest that higher muscle damage (reflected by elevated CPK) is associated with poorer motor function in children with Duchenne Muscular Dystrophy.

Table 6: Pairwise comparisons of CPK between MMT categories (Mann-Whitney U; Bonferroni adjusted)

Group 1	Group 2	U statistic	p value	Bonferroni-adjusted p
Low	Moderate	212.0	0.0065	0.0196
Low	High	221.5	0.0022	0.0066
Moderate	High	244.0	0.0006	0.0019

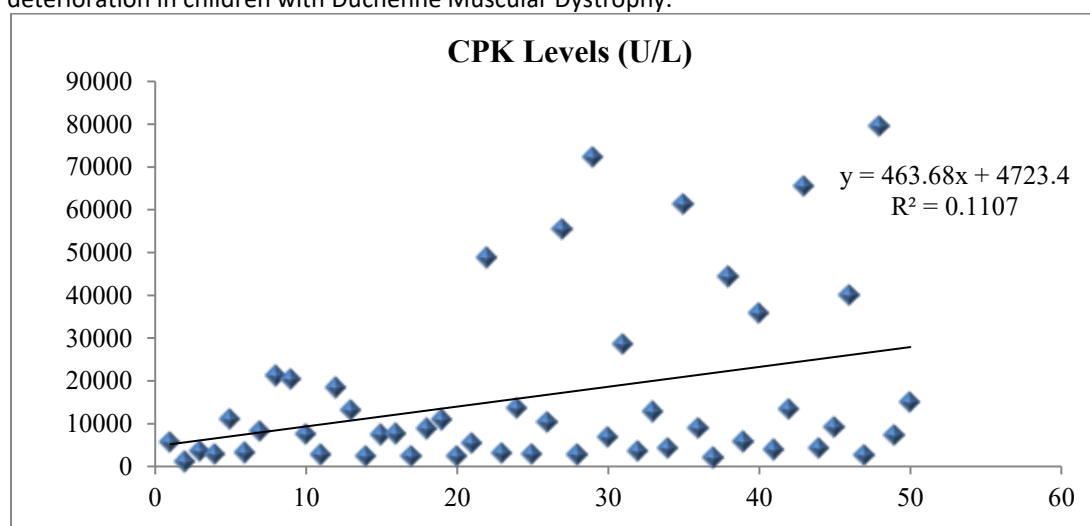
The pairwise comparison of serum CPK levels between different MMT categories using the Mann-Whitney U test with Bonferroni adjustment. The results show that there was a statistically significant difference in CPK levels between all groups. Specifically, significant differences were observed between low vs moderate (adjusted p = 0.0196), low vs high (adjusted p = 0.0066) and moderate vs high (adjusted p = 0.0019) MMT categories. This confirms that each level of muscle strength is distinctly associated with different levels of muscle enzyme elevation. The findings reinforce that serum CPK is a sensitive biomarker that significantly varies with disease severity and muscle function.

Table 7: Association between serum CPK and motor function (correlation tests)

Motor function measure	Spearman ρ	p-value	Pearson r (log10)	p-value
Neck flexors	-0.636	0.0000*	-0.653	0.0000*
Shoulder abductors	-0.609	0.0000*	-0.640	0.0000*
Knee extensors	-0.577	0.0000*	-0.582	0.0000*
Average MMT	-0.633	0.0000*	-0.654	0.0000*

*Correlation is significant at the 0.01 level (2-tailed)

The correlation between serum CPK levels and various motor function parameters using both Spearman's correlation coefficient (ρ) and Pearson's correlation coefficient (r). A strong negative correlation was observed between CPK levels and all motor function measures including neck flexors ($\rho = -0.636$), shoulder abductors ($\rho = -0.609$), knee extensors ($\rho = -0.577$) and average MMT score ($\rho = -0.633$). Similar negative correlations were also noted with Pearson's test after logarithmic transformation. All correlations were found to be highly statistically significant ($p < 0.001$). This indicates that as serum CPK levels increase, muscle strength significantly decreases. Thus, serum CPK serves as a reliable indicator of disease severity and motor function deterioration in children with Duchenne Muscular Dystrophy.



DISCUSSION

The present study included 50 children diagnosed with DMD and aimed to correlate serum CPK levels with motor function assessed by MMT grading of neck flexors, shoulder abductors and knee extensors. The findings are discussed with published literature, focusing on motor impairment, CPK variation across functional groups and the relationship between biochemical and functional markers. The mean age of the study population was 9.68 ± 3.285 years, with a median age of 9.00 years. Most children were aged 5–10 years, 27 (54%), followed by 11–15 years, 19 (38%), below 5 years, 3 (6%), and above 15 years, 1 (2%). This shows that DMD is mainly evaluated during school age and early adolescence. Kim et al. (2017)³ reported maximum serum CK activity between 2–5 years, while Sadek et al. (2017)¹¹, Zhang et al. (2015)¹², Wang et al. (2017)¹³, Luiz et al. (2019)¹⁴ and Degan et al. (2025)¹⁵ also reported pediatric and adolescent age ranges comparable to DMD progression. Hashim et al. (2011)¹⁶ described onset at 2–3 years, which supports the early disease group in our cohort.

The study showed a significant positive relationship between motor function and Global Development Quotient (GDQ). Mean GDQ increased from 42.94% in the Low MMT group to 57.59% in the Moderate group and 71.82% in the High group ($F = 18.791$; $p = 0.001$). This suggests that poorer motor function is associated with greater developmental impairment. Pane et al. (2013)¹⁷ reported borderline or low developmental quotients in boys with DMD, while Connolly et al. (2013)¹⁸ found significantly reduced motor scores, especially gross motor function. Cyrulnik et al. (2008)¹⁹ reported impairments in language, fine motor, attention and memory domains. Rasic et al. (2015)²⁰, Chamova et al. (2013)²¹ and Goyal et al. (2021)²² also reported cognitive and developmental involvement in DMD patients.

Clinical Features by MMT Category

Gower's Sign: In the present study, Gower's sign was present in 78% of all children, with the highest prevalence in the Low MMT group (100%) and lower prevalence in the High MMT group (41.2%). This is similar to Goyal et al. (2021)²², who reported Gower's sign in 85.3% of Indian DMD patients. Sattenapalli et al. (2022)²³ documented Gower's sign as universally present in ambulatory DMD patients, while Sarkar et al. (2023)²⁴ reported it in 86% of their cohort. The decline across MMT categories reflects progressive hip extensor and gluteal weakness.

Waddling Gait: Waddling gait was present in 75% of Low, 76.5% of Moderate and 17.6% of High MMT children, showing a significant association ($\chi^2 = 15.384$; $p = 0.000$). This finding is consistent with Venugopal et al. (2023)²⁵, who described waddling gait as a characteristic early feature of DMD caused by proximal pelvic girdle weakness. Goyal et al. (2021)²² also reported gait abnormalities in ambulatory DMD boys. The low prevalence in the High MMT group suggests that waddling gait is more common in moderate-to-severe motor impairment.

Toe Walking: Toe walking was observed in 93.8% of Low, 52.9% of Moderate and 58.8% of High MMT children ($\chi^2 = 7.305$; $p = 0.026$). Although Sarkar et al. (2023)²⁴ reported toe walking in only 11% of their cohort, Goyal et al. (2021)²² reported it in 44.8% of Indian DMD children. Venugopal et al. (2023)²⁵ described toe walking as an early ambulatory-stage feature due to Achilles tendon shortening and calf tightness, explaining its persistence even in children with relatively preserved MMT scores.

Supine to Sitting: Difficulty in supine to sitting was found in 81.3% of Low, 47.1% of Moderate and 17.6% of High MMT children ($\chi^2 = 13.368$; $p = 0.001$). This pattern is supported by Baranello et al. (2024)²⁶, who identified timed rise from floor as a sensitive marker of motor deterioration and earlier loss of ambulation in DMD. Bushby et al. (2010)²⁷ and TREAT-NMD care guidelines also describe difficulty rising from the floor as an early functional loss reflecting proximal hip and knee extensor weakness.

Hypertrophied Muscles: Hypertrophied muscles were observed in 68.8% of Low, 64.7% of Moderate and 82.4% of High MMT children, with no significant difference across groups ($\chi^2 = 1.436$; $p = 0.488$). Kornegay et al. (2012)²⁸ reported calf enlargement in 94% of Indian DMD patients, highlighting pseudohypertrophy as a common and early feature. Droracle (2026)²⁹ also confirmed calf pseudohypertrophy in 94% of DMD patients. Similarly, Goyal et al. (2021)²² reported calf hypertrophy in 91.3% of their cohort, showing that muscle hypertrophy may occur independently of functional severity.

Serum CPK Levels by MMT Category

Serum CPK levels were markedly elevated across all MMT categories in the present study, confirming the characteristic biochemical profile of DMD. The overall CPK range was 1,070–79,456 U/L, far above the normal pediatric range of 10–170 U/L and consistent with reports that CPK in DMD may be 50–200 times normal. Sun et al. (2008)³⁰ reported CK levels ranging from 2,595–45,495 U/L in 40 DMD children aged 3–14 years, which is broadly comparable to our cohort. The Low MMT group showed the highest CPK levels, with a mean of $36,314.31 \pm 26,157.72$ U/L and median of 37,832 U/L, suggesting greater biochemical evidence of muscle destruction in children with severe motor impairment. Sun et al. (2008)³⁰ reported peak CK levels of 27,750–31,173 U/L in children aged 3–5 years, the period of maximal muscle necrosis. Awano et al. (2024)² similarly found that CK levels remain extremely high until age 6 years and decline rapidly from ages 7–12 years, corresponding with worsening motor decline.

A clear inverse gradient was observed, with CPK decreasing as MMT category improved: Low MMT mean 36,314 U/L, Moderate MMT mean 9,459.82 U/L and High MMT mean 5,030.06 U/L. Burch et al. (2015)³¹ reported a mean serum CK of $7,443 \pm 6,357$ U/L in a mixed-age DMD cohort, which is close to our Moderate MMT group. Awano et al. (2024)² also confirmed that CK and related biomarkers decrease with declining motor function, supporting the pattern seen in our MMT stratification. The Kruskal-Wallis test showed a significant difference in CPK distribution across MMT categories ($H = 17.299$; $p = 0.0001$). Pairwise Mann-Whitney U comparisons remained significant after Bonferroni correction, with Moderate vs. High showing the strongest significance ($p = 0.0019$), followed by Low vs. High ($p = 0.0066$) and Low vs. Moderate ($p = 0.0196$). The use of non-parametric testing with Bonferroni correction is appropriate because CPK values in DMD are highly skewed, consistent with Diniz et al. (2012)³² and other DMD biomarker studies.

The significant difference between Moderate and High MMT groups suggests that CPK is not only a diagnostic marker but may also reflect graded muscle damage within ambulatory functional stages. This supports the view of Kim et al. (2017)³, who found CK to be an important correlate of functional outcomes even in children who retained ambulation.

CONCLUSION

The present study demonstrates a clear relationship between serum creatine kinase (CPK) levels and motor function in children with Duchenne Muscular Dystrophy. The majority of patients presented in mid-childhood, with a mean age of approximately 9.7 years, highlighting the typical age of clinical manifestation. Motor function, as assessed by Manual Muscle Testing (MMT), showed moderate weakness overall, with relatively greater involvement of lower limb muscles. A significant association was observed between motor function and global developmental status, with higher MMT scores corresponding to better developmental quotients. Clinical features such as Gower's sign, waddling gait, toe walking, and difficulty in rising from supine position were more prevalent in patients with poorer motor strength, reinforcing their utility as indicators of disease severity. Serum CPK levels were markedly elevated in patients with lower MMT scores and showed a progressive decline with improvement in motor function. Statistical analysis confirmed significant differences in CPK levels across all MMT categories, along with a strong negative correlation between CPK and muscle strength. This indicates that higher enzyme levels are associated with greater muscle damage and poorer functional status.

REFERENCES

1. Zatz M, Rapaport D, Vainzof M, Passos-Bueno MR, Bortolini ER, Pavanello RD, Peres CA. Serum creatine-kinase (CK) and pyruvate-kinase (PK) activities in Duchenne (DMD) as compared with Becker (BMD) muscular dystrophy. *Journal of the neurological sciences*. 1991 Apr 1;102(2):190-6.
2. Awano H, Nambu Y, Itoh C, Kida A, Yamamoto T, Lee T, Takeshima Y, Nozu K, Matsuo M. Longitudinal data of serum creatine kinase levels and motor, pulmonary, and cardiac functions in 337 patients with Duchenne muscular dystrophy. *Muscle & Nerve*. 2024 May;69(5):604-12.
3. Kim EY, Lee JW, Suh MR, Choi WA, Kang SW, Oh HJ. Correlation of serum creatine kinase level with pulmonary function in duchenne muscular dystrophy. *Annals of rehabilitation medicine*. 2017 Apr 27;41(2):306-12.
4. Shao J, Wan B, Gao T, Shi Y, Zhu Y, Yu XE. Hydrogen-rich water improves skeletal muscle damage in mice with Duchenne muscular dystrophy.
5. Rohlenová M, Machová K, Baranová J, Mokrá L, Mensová L, Mazanec R, Juříková L, Staněk J, Fuchsová P, Lauerová B, Kumhera M. Serum Creatine Kinase and Transaminase Levels in Duchenne and Becker Muscular Dystrophies. *Muscle & Nerve*. 2025 May 11.
6. Nambu Y, Osawa K, Shirakawa T, Sunami A, Sonehara S, Bo R, Nozu K, Matsuo M, Awano H. Serum titin/creatinine ratio as a biomarker for discriminating disease severity in Duchenne and Becker muscular dystrophies. *Frontiers in Neurology*. 2025 Jul 9;16:1591748.
7. Konagaya M, Takayanagi T. Regularity in the Change of Serum Creatine Kinase Level in Duchenne Muscular Dystrophy-A Study with Long-ter Follow-up Cases. *Japanese journal of medicine*. 1986;25(1):2-8.
8. Tarnopolsky MA, Mahoney DJ, Vajsar J, Rodriguez C, Doherty TJ, Roy BD, Biggar D. Creatine monohydrate enhances strength and body composition in Duchenne muscular dystrophy. *Neurology*. 2004 May 25;62(10):1771-7.
9. Bellis MD, Imbrici P, Lenti R, Liantonio A, De Luca A. State-of-the-Art Research and New Pharmacological Perspectives on Renal Involvement in Duchenne Muscular Dystrophy: A Narrative Review. *Biomedicines*. 2026 Jan 21;14(1):230.
10. Mah JK, Korngut L, Dykeman J, Day L, Pringsheim T, Jette N. A systematic review and meta-analysis on the epidemiology of Duchenne and Becker muscular dystrophy. *Neuromuscular Disorders*. 2014 Jun 1;24(6):482-91.
11. Sadek AA, Mahmoud SM, Abd El-Aal M, Allam AA, Abd El-Halim WI. Evaluation of cardiac functions in children with Duchenne muscular dystrophy: a prospective case-control study. *Electronic physician*. 2017 Nov 25;9(11):5732.
12. Zhang H, Zhu Y, Sun Y, Liang Y, Li Y, Zhang Y, Deng L, Wen X, Zhang C. Serum creatinine level: a supplemental index to distinguish Duchenne muscular dystrophy from Becker muscular dystrophy. *Disease markers*. 2015;2015(1):141856.
13. Wang L, Chen M, He R, Sun Y, Yang J, Xiao L, Cao J, Zhang H, Zhang C. Serum creatinine distinguishes Duchenne muscular dystrophy from Becker muscular dystrophy in patients aged ≤ 3 years: a retrospective study. *Frontiers in Neurology*. 2017 May 8;8:196.
14. Luiz LC, Marson FA, Almeida CC, Toro AA, Nucci A, Ribeiro JD. Analysis of motor and respiratory function in Duchenne muscular dystrophy patients. *Respiratory physiology & neurobiology*. 2019 Apr 1;262:1-1.

15. Degan C, Tsonaka R, de Vries SI, Ikelaar N, van der Holst M, Kan HE, Niks EH, Spitali P. Creatine/creatinine ratio and myostatin as biomarkers to monitor muscle function in Duchenne Muscular Dystrophy patients. medRxiv. 2025 Aug 14.
16. Hashim R, Shaheen S, Ahmad S, Sattar A, Khan FA. Comparison of serum creatine kinase estimation with short tandem repeats based linkage analysis in carriers and affected children of duchenne muscular dystrophy. Journal of Ayub Medical College Abbottabad. 2011 Mar 1;23(1):125-8.
17. Pane M, Scalise R, Berardinelli A, D'Angelo G, Ricotti V, Alfieri P, Moroni I, Hartley L, Pera MC, Baranello G, Catteruccia M. Early neurodevelopmental assessment in Duchenne muscular dystrophy. Neuromuscular Disorders. 2013 Jun 1;23(6):451-5.
18. Connolly AM, Florence JM, Craddock MM, Malkus EC, Schierbecker JR, Siener CA, Wulf CO, Anand P, Golumbek PT, Zaidman CM, Miller JP. Motor and cognitive assessment of infants and young boys with Duchenne Muscular Dystrophy: results from the Muscular Dystrophy Association DMD Clinical Research Network. Neuromuscular Disorders. 2013 Jul 1;23(7):529-39.
19. Cyrulnik SE, Fee RJ, Batchelder A, Kiefel J, Goldstein E, Hinton VJ. Cognitive and adaptive deficits in young children with Duchenne muscular dystrophy (DMD). Journal of the International Neuropsychological Society. 2008 Sep;14(5):853-61.
20. Rasic VM, Vojinovic D, Pesovic J, Mijalkovic G, Lukic V, Mladenovic J, Kosac A, Novakovic I, Maksimovic N, Romac S, Todorovic S. Intellectual ability in the duchenne muscular dystrophy and dystrophin gene mutation location. Balkan Journal of Medical Genetics. 2015 Apr 10;17(2):25.
21. Chamova T, Guergueltcheva V, Raycheva M, Todorov T, Genova J, Bichev S, Bojinova V, Mitev V, Tournev I, Todorova A. Association between loss of dp140 and cognitive impairment in duchenne and becker dystrophies. Balkan Journal of Medical Genetics. 2013 Oct 3;16(1):21-9.
22. Goyal M, Gupta A, Agarwal K, Kapoor S, Kumar S. Duchenne muscular dystrophy: genetic and clinical profile in the population of Rajasthan, India. Annals of Indian Academy of Neurology. 2021 Nov 1;24(6):873-8.
23. Sattenapalli NC, Areti AR, Kulandaivelu US, Alavala RR, Manne R. Study of Clinical Features and Diagnosis Pattern of Duchene Muscular Dystrophy in Southern India. Journal of Neurosciences in Rural Practice. 2022 Jan 5;13(1):43.
24. Sarker S, Eshaque TB, Soorajkumar A, Nassir N, Zehra B, Kanta SI, Rahaman MA, Islam A, Akter S, Ali MK, Mim RA. Mutational spectrum and phenotypic variability of Duchenne muscular dystrophy and related disorders in a Bangladeshi population. Scientific Reports. 2023 Dec 6;13(1):21547.
25. Venugopal V, Pavlakis S. Duchenne muscular dystrophy. InStatPearls [Internet] 2023 Jul 10. StatPearls Publishing.
26. Baranello G, Muntoni F. AAV gene therapy for Duchenne Muscular Dystrophy: lessons learned from a phase 3 trial. Gene Therapy. 2024 Nov;31(11):541-3.
27. Bushby K, Finkel R, Birnkrant DJ, Case LE, Clemens PR, Cripe L, Kaul A, Kinnett K, McDonald C, Pandya S, Poysky J. Diagnosis and management of Duchenne muscular dystrophy, part 2: implementation of multidisciplinary care. The Lancet Neurology. 2010 Feb 1;9(2):177-89.
28. Kornegay JN, Childers MK, Bogan DJ, Bogan JR, Nghiem P, Wang J, Fan Z, Howard Jr JF, Schatzberg SJ, Dow JL, Grange RW. The paradox of muscle hypertrophy in muscular dystrophy. Physical medicine and rehabilitation clinics of North America. 2012 Feb;23(1):149.
29. <https://www.droracle.ai/articles/751819/what-is-pseudohypertrophy-in-duchenne-muscular-dystrophy-dmd-in>
30. Sun SC, Peng YS, He JB. Changes of serum creatine kinase levels in children with Duchenne muscular dystrophy. Zhongguo dang dai er ke za zhi= Chinese journal of contemporary pediatrics. 2008 Feb 1;10(1):35-7.
31. Burch PM, Pogoryelova O, Goldstein R, Bennett D, Guglieri M, Straub V, Bushby K, Lochmüller H, Morris C. Muscle-derived proteins as serum biomarkers for monitoring disease progression in three forms of muscular dystrophy. Journal of neuromuscular diseases. 2015 Sep 2;2(3):241-55.
32. Diniz GP, Lasmar LM, Giannetti JG. Motor assessment in patients with Duchenne muscular dystrophy. Arquivos de neuro-psiquiatria. 2012 Jun 11;70(6):416-21.