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Original Research Article

MOTOR NERVE CONDUCTION STUDY IN CHILDREN WITH SEVERE ACUTE MALNUTRITION UNDER 5 YEARS OF AGE

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Abstract

Background: Severe acute malnourishment (SAM) is a significant public health problem in India and many developing countries. Malnutrition slows down the myelination process, thus preventing the increase in the calibre of myelinated nerve fibres. Motor nerve conduction changes caused by severe acute malnutrition can be evaluated clinically and electrophysiologically. These changes are produced mainly due to a deficiency of micro and macronutrients like vitamins, minerals, protein, fat & Carbohydrates. This study aimed to assess the effects of severe acute malnutrition on children's peripheral motor median, ulnar, tibial, and peroneal nerve conduction velocities, distal latencies, and amplitudes. **Materials and Methods:** The study group included 50 severely acute malnourished children (SAM) of 6-59 months of age, recruited from the severe malnutrition treatment unit (SMTU), J.A. Group of the Hospital, G.R.M.C. Gwalior (M.P.), based on WHO classification for severe acute malnutrition. The Control group consisted of 50 normally nourished healthy children of the same age group. The nerve conduction study for the peripheral motor nerves was performed using the four-channel RMS - EMG ES MARK- II machine. Results were analyzed statistically using the unpaired student's t-test. **Result:** A nerve conduction study (NCS) demonstrated reduced motor nerve conduction velocity ($p < 0.05$) and delayed distal latencies in the median and ulnar motor nerves of both upper extremities, the tibial and peroneal motor nerves of lower extremities in children with severe acute (SAM) malnutrition. **Conclusion:** The present study shows a statistically significant decrease in nerve conduction velocity in children with severe acute malnutrition. In our study, distal latencies were prolonged, but no significant changes were seen in the median and ulnar motor nerve amplitude.

INTRODUCTION

Nutrition has a strong influence on maintaining health and preventing disease. The nutritional status of any country's children is a good window into its development.[1] Undernutrition (including intrauterine growth restriction (IUGR), protein energy malnutrition (PEM), stunting, wasting, and micro and macronutrient deficiencies) contributes to 45 % of total child deaths worldwide. Severe acute malnutrition is children's most severe and life-threatening form of malnutrition. The NFHS (2006) estimates the number of malnourished children in this central Indian state (Madhya Pradesh) at a whopping 6 million, which is more than 60 percent of the total number of children under 5 years of age. Out of these 6 million malnourished children, 1.3 million have Severe Acute Malnutrition (SAM).[2] According to the latest NFHS-5 survey (2019–2021),[3] covering 36 states and union territories (UTs), the prevalence remains at an alarming 7.7%. SAM is defined as weight-for-height below the −3 z-scores of the median, whose growth standards are known as severe wasting.[4] Children with SAM have a 9–11 times higher risk of mortality and morbidity than well-nourished children.[5,6] Nutrition plays a key role in children's physical and mental development, especially in the formative years of life. Due to urbanization and industrialization, the practice of breastfeeding has declined, and

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malnutrition appears at an earlier age. This factor is of prime importance because it is a period of rapid growth and development (Stoch and Smythe, 1976).[7] Various animal and human studies have shown that malnutrition as an isolated entity causes physical, chemical, and functional changes in the brain (Dobbing and Widdowson, 1965; Cravioto et al., 1966; Chase et al., 1971).[8,9] During the development of the human nervous system, myelination begins in the motor roots of the peripheral nervous system and is followed by myelination of the spinal cord and brain. Most myelin is assembled during the first years of postnatal life.[10] Myelination of the Peripheral nerve begins about the 15th week of gestation and continues during the first 2 to 5 years of life.[11-13])Malnutrition in children under five can affect the process of myelination.[14] The loss of myelin may delay or block conduction in the demyelinated axons. Nerve conduction tests can detect slow conduction in peripheral nerves by measuring nerve conduction velocity. This study relates to the predictable peripheral nerve responses in children suffering from severe acute malnutrition (SAM).

MATERIALS AND METHODS

The present study is a cross-sectional case-control study. The study was conducted in the postgraduate Laboratory, Department of Physiology, Gajra Raja Medical College and J.A. Group of Hospital, Gwalior (M.P.). Institutional Ethical Committee approval was obtained before commencing the study. Informed written consent was taken from the Parents/guardians of the children. The study group included 50 severely acute malnourished children (SAM) of 6–59 months of age, recruited from the severe malnutrition treatment unit (SMTU), J.A. Group of the Hospital, G.R.M.C. Gwalior (M.P.), based on WHO classification for severe acute malnutrition. The Control group consisted of 50 normally nourished healthy children of the same age group. The case-control study period was extended from January 2021 to December 2021. The family, immunization, birth, metabolic, endocrinal disorder, and history of neuromuscular diseases were taken in detail. Assessment of nutritional status was done according to the guidelines of the WHO and the Indian Academy of Paediatrics.[15] The nerve conduction study for the motor median, ulnar (both upper limb) nerves and motor tibial, peroneal (both lower limb) was performed by using the four-channel RMS - EMG ES MARK- II machine. Nerves were stimulated by applying depolarising square wave electrical pulses to the skin over the peripheral nerves. The surface recording electrodes were used to measure Motor Nerve Conduction Velocity (MNCV). The ground electrode was placed on the dorsum of the hand between stimulating and active electrodes. The active electrodes were placed over the midpoint of the belly of the abductor pollicis Brevis muscle for the Median nerve and the abductor digiti minimi muscle for the Ulnar nerve. The reference electrode was placed 3 cm distally over the 1st metacarpophalangeal joint for the Median nerve and the 5th metacarpophalangeal joint for the Ulnar nerve. A supra maximal stimulations were given at the wrist (3 cm proximal to the distal crease) and the elbow (near the volar crease of the brachial pulse) for the Median nerve. Ulnar stimulation was given at the wrist 3 cm distal to the crease and elbow 3 to 4 cm distal to the medial epicondyle at 135 degrees of flexion. Peroneal nerve action potential was recorded orthodromically with an active electrode placed over the belly of the Extensor Digitorum Brevis (EDB) muscle. The reference electrode was placed distally over the lateral aspect of the 5th metatarsal head. The ground electrode was placed on the dorsum of the foot. Peroneal nerve stimulation was delivered at the fibular neck.

The Tibial nerve action potential was recorded with the active electrode placed over the belly of the abductor hollicus muscle, 1 cm inferior and 1cm proximal to the navicular prominence. The reference electrode was placed distally over the medial aspect of 1st metatarsal head. The ground electrode was placed over the dorsum of the foot. Distal stimulation was delivered 10 cm proximal to the active electrode and posterior to the medial malleolus. Proximal stimulation was delivered to the Tibial nerve in the popliteal fossa. The distance between the points of stimulation was measured and the response to the stimuli at the corresponding muscles was recorded by the surface electrode. The latency of the first response was subtracted from the latency of the second response thus the time taken for the impulse to travel from the first to the second point of stimulation in each nerve was obtained. Nerve conduction study parameters were evaluated for velocity, distal latency, and amplitudes. Results were analyzed statistically using the unpaired student's t-test. Windows Software SPSS 20 was used for the statistical analysis. A p-value of less than 0.05 was taken as statistically significant.

RESULTS

We included 20 (40.0%) females and 30 (60.0%) males as a case in our study, with the same distribution in control. The anthropometrical values of SAM were significantly different from the control. [Tables 2 & 3] depict that Mean distal latency and velocities of the median and ulnar nerve was significantly reduced in children with severe acute malnutrition ($p < 0.01$). There was no significant change in the amplitudes of both group. In the nerves of the lower extremities, the conduction velocities were significantly reduced in the SAM group [Tables 3 & 4].

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Table 1: Anthropometric comparison in case and control

Anthropometry	Severe acute Malnourished Mean ± SD	Normal(control) Mean ± SD	t (P) value
Height (cm)	76.76 ± 9.76	90.06±10.97	6.40 (0.01*)
Weight (kg)	7.33 ±1.38	12.28±2.00	14.40 (0.01*)
MUAC (cm)	10.04±0.98	12.62±0.53	16.30 (0.01*)
Age (years)	2.73±1.30	2.83±1.23	0.39 (0.701)
* Significant Statistically			

Table 2: Comparison of Median motor nerve conduction parameters in case and control

Median motor nerve conduction parameters		Severe acute malnourished Mean ± SD	Normal Mean ± SD	t (p) value
Latency(msec)	L	3.27±1.7	2.19±0.7	4.26 (0.01*)
	R	2.66±0.9	1.85±0.6	5.50 (0.01*)
Amplitude(mv)	L	3.68±2.1	4.34±2.0	-1.60 (0.122)
	R	3.91±2.3	4.60±1.9	-1.61 (0.11)
Velocity(m/s)	L	34.31±12.3	47.70±18.3	-4.29 (0.01*)
	R	34.39±16.7	42.10±14.6	-2.46 (0.01*)
* Significant Statistically L=Left, R-Right, msec=Mili second, mv-Mili Volt, m/s = Meter per second				

Table 3: Comparison of Ulnar motor nerve parameters in case and control

Ulnar motor nerve conduction parameters		Severe acute malnourished Mean ± SD	Normal nourished Mean ± SD	t (p) value
Latency (msec)	L	2.52±0.3	2.29±0.4	2.90 (0.005*)
	R	2.42±0.3	2.18±0.4	3.223 (0.002*)
Amplitude (mv)	L	3.51±0.9	4.14±1.1	-3.047 (0.003*)
	R	3.45±1.2	3.95±1.1	-2.196 (0.030*)
Velocity (m/s)	L	35.96±5.8	39.96±3.2	-4.281 (0.01*)
	R	39.49±8.5	44.54±5.9	-3.433 (0.001*)
* Significant Statistically				

Table 4: Comparison of Tibial motor nerve parameters in case and control

Tibial motor nerve parameters		Severe Acute Malnourished Mean ± SD	Normal nourished Mean ± SD	t (p) value
Latency (msec)	L	2.53±0.7	2.13±0.6	3.139 (0.002*)
	R	2.53±0.5	2.20±0.4	3.615 (0.001*)
Amplitude (mv)	L	4.16±0.7	4.93±1.1	-4.075 (0.001*)
	R	4.34±0.7	4.99±1.3	-3.120 (0.002*)
Velocity (m/s)	L	32.83±4.1	34.73±2.6	-2.740 (0.007*)
	R	32.41±3.69	35.26±3.14	-4.153 (0.001*)
* Significant Statistically				

Table 5: Comparison of Peroneal Motor Nerve parameters in case and control

Peroneal Motor Nerve parameters		Severe Acute Malnourished Mean ± SD	Normal nourished Mean ± SD	t (p) value
Latency (msec)	L	3.15±0.6	2.90±0.5	2.085 (0.040*)
	R	2.99±0.5	2.44±0.5	5.259 (0.001*)
Amplitude (mv)	L	4.09±0.9	4.69±1.1	-2.907 (0.005*)
	R	4.12±1.0	4.77±1.3	-2.819 (0.006*)
Velocity (m/s)	L	34.52±5.1	40.41±5.9	-5.301 (0.001*)
	R	33.65±4.1	40.74±4.6	-8.154 (0.001*)
* Significant Statistically				

DISCUSSION

Maturation of the peripheral nervous system in the form of myelination begins during the fourth month of fetal life and it is complete at around 5 years of age.[16] Myelination, the diameter of the nerve fiber, and internodal differences are the determinants of nerve conduction velocity. As the majority of myelin is assembled during the first years of postnatal life, a great magnitude of tasks is performed by glial cells during this short period.[17] Ghosh S et al,[18] they reported a significant reduction in motor nerve conduction velocity in children with severe PEM and ongoing long-term malnutrition. Osuntokun BO,[19]reported that motor nerve conduction velocity was decreased to the greatest extent in the more severe cases of PEM and the slowest mean rate was that in 7 patients who subsequently died. Chopra et al,[20] studied sural nerve biopsies and reported significant segmental demyelination in about 50 percent of cases in a group consisting of children with severe PEM.

In our study, the difference between the conduction velocities was significantly reduced in severe acute malnutrition. There was a delay in the distal latencies in the SAM group. The nerves of the lower limbs were relatively more compromised as opposed to the upper limb nerves. This may be because the process of myelination lasts longer in the long

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nerves of the lower extremities, thus making them more vulnerable to the insults of postnatal malnutrition. Similarly, Sachdeva, Taori, and Pereira,[21] reported delayed nerve conduction velocities in 12 kwashiorkor children between the ages of one to four years.

CONCLUSION

Severe acute malnutrition affects the maturation of peripheral nerves as indicated in the present study by a significant decrease in motor nerve conduction velocity in severely malnourished children. It indicates an association between the severity of malnutrition with changes in nerve conduction. The detrimental effects of malnutrition may be the cause of long-term neurological deficits in children with PEM.

Limitation of our study

Follow-up studies should assess children who have recovered from malnutrition. Subclinical deficiencies cannot be ruled out. While this study had many strengths, it was limited by its biases.

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