Peripheral and central nerve conduction changes in protein energy malnutrition: Relation to Cyanocobalamine status

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ABSTRACT

Background: Peripheral and central nervous system (CNS) injuries caused by severe malnutrition can be shown clinically and electrophysiologically. They are produced mainly due to the accompanying multiple macro and micro nutrient deficiencies. Design: Upper and lower limbs nerve conduction velocities (NCV) and somatosensory evoked potentials (SSEP) at the Erb’s point (N₉), the lower cervical area (N₁₃) and the contralateral sensory cortex (N₂₀) as well as the distances between them, were assessed in 20 infants having protein energy malnutrition (PEM), who were subdivided into two groups, ten non-edematous and ten edematous patients. They were compared to a cohort of ten healthy infants. Besides the anthropometric measurements and usual laboratory investigations, Cyanocobalamine (vitamin B₁₂) level was assessed in all the studied infants. These measures were repeated after two months ± two weeks of nutritional rehabilitation of the malnourished infants. Results: The current study revealed significant delay in central rather than peripheral nerve conduction (especially at N₁₃, N₉-₁₃ and N₉-₂₀) coupled by significant decrease in vitamin B₁₂ levels in both types of PEM compared to the controls. There was significant improvement in each of these studied parameters after nutritional rehabilitation yet the levels did not reach the control values. Conclusion: In conclusion, PEM disregards its type, cause disturbed central nerve conduction more than peripheral and this abnormality is reversible with early and optimum nutritional rehabilitation. Vitamin B₁₂ is partially responsible for this delay and the improvement of its level on nutritional rehabilitation couples the improvement in nerve myelination abnormalities. We thus recommend early detection and proper management of cases of malnutrition among infants with long periods of follow up and special emphasis on providing the necessary neurotropic elements, as this age has proved to be very vulnerable to neurological sequelae resulting from such deficiencies. (Egypt J. Neurol. Psychiat. Neurosurg., 2004, 41(1): 359-371).

INTRODUCTION

There are changes in peripheral nerve conduction velocity and brain stem evoked potentials in children with marasmus and Kwashiorkor, the most severe forms of acute malnutrition¹⁻³. There is evidence that myelination process is impaired in malnourished children and animals⁴. CNS injuries caused by severe malnutrition can be shown clinically and electrophysiologically⁵ and measurement of SSEP is a standard clinical technique for assessing the function of peripheral tracts and their central projections⁵. In addition, among the neurotropic factors reported to be deficient in PEM is vitamin B₁₂⁷. Although there is some improvement in the prevalence of underweight and stunting in some regions of the world over the past two decades, the population of the developing world increased during this time; thus, the total number
of underweight and stunted children has not changed dramatically since 1980. Approximately one-third of the child population suffers from PEM worldwide, affecting mainly developing countries. Researchers are thus still concerned with the problems caused by the acute forms of under nutrition and malnutrition.

This study was thus designed to assess the NCV and SSEP in PEM patients and correlate any defect detected to the level of vitamin B₁₂ in such patients with special emphasis on the effect of nutritional rehabilitation.

**PATIENTS AND METHODS**

Twenty infants with PEM according to Wellcome were recruited from the Inpatient Department of Children’s Hospital, Ain Shams University, Cairo, Egypt in the period from August 2002 till January 2003. They were further divided into edematous and non-edematous groups according to the presence and absence of edema and their mean weight for age percent. The patients were 10 males and 10 females with an age range of 6 to 16 months. They were compared to a cohort of ten healthy infants (5 males and 5 females with an age range of 6-18 months). All the studied cases were from low socioeconomic standard families according to Park and Park.

After obtaining the approval of the ethical committee of the Children’s Hospital, Ain Shams University, a written consent was taken from the parents or legal guardians. Each of the studied infants was enrolled in a 3 phase study with pre-interventional assessment as phase I, nutritional rehabilitation interventional program as phase II and post-interventional assessment two months ± two weeks from the start (phase III).

Nutritional rehabilitation was done according to the WHO. Initially, management of life threatening and emergency conditions were done in the first week then the start of feeding was supervised. Caloric intake was 80-100 Kcal/kg/day with the continuity of breast-feeding in cases of breast fed infants. The diet given was low in protein, fat, and sodium, high in carbohydrates as almost all severely malnourished infants have infections, impaired liver and intestinal functions and problems related to electrolyte imbalance.

Rehabilitation stage followed with the return of the infant’s appetite. The caloric intake increased to 150-200 Kcal/Kg/day with increase in the amounts and decrease in the frequency. High protein diet was given and vitamins and minerals (potassium, magnesium and zinc) were continued in increased amounts. Iron was given during this stage to treat the anemia present. The infant remained in the hospital for the first part of this rehabilitation phase (at least 3 weeks after admission), and then followed up in the nutritional rehabilitation outpatient clinic.

Assessment in phases I and III included detailed dietetic history (using a questionnaire answered by the parents or the care giver laying stress on the 24 hour recall of food given to the infants) and clinical examination with special emphasis on the anthropometric measurements and signs of malnutrition. The laboratory workup comprised complete blood count (Coulter T660, Miami, USA), serum albumin, liver and kidney functions (autoanalyzer, Synchron CX-5 delta of Beckman) as well as assessment of vitamin B₁₂ in the serum by radioimmunoassay according to Mollin et al. and Gilbert and Mailhot. Patients’ assessment also included NCV study of peripheral nerve myelination function represented by upper limbs and lower limbs distal latency and conduction velocity, and central sensory conduction represented by SSEP in malnourished children and control group to assess the effects of acute malnutrition on central and peripheral nervous conduction. NCV was recorded by surface electrodes placed on the corresponding muscle using the belly-tendon method according to Gamstorp and Shelburne. SSEPs were measured at three locations along the sensory pathway of the median nerve and these were the Erb’s point, the lower cervical area and the...
contralateral sensory cortex. By using this method, the interwave latency can be measured and any central dysfunction can be differentiated from a peripheral one.

Statistical analysis of the results was done via the standard computer programs SPSS (version 10) and Statistica software package version 5 (Statsoft, Tulsa, OK, USA). Non-parametric data were detected by the Shapiro-Wilk test. Student-t and Paired-t tests were used for parametric quantitative data and Mann-Whitney U and Wilcoxon matched pairs tests for non-parametric quantitative data in addition to the Correlation studies. The numerical data were represented in mean±SD and median (interquartile range). The differences were considered significant if the probability (p) values were less than 0.05.

**RESULTS**

The present study revealed significantly lower anthropometric measurements in PEM patients compared to those of the controls and these measurements showed significant improvement after nutritional rehabilitation yet not reaching the control values (tables 1 and 2). The same findings were observed regarding hemoglobin level and serum albumin (tables 1 and 2). As regards liver and kidney functions, they were within the normal range for age and sex according to Nicholson and Pesce, from the start of the study.

Vitamin B₁₂ values were significantly lower in both studied PEM subgroups compared to those of the controls and showed significant improvement after nutritional rehabilitation but again its values did not reach that of the controls (tables 1 and 2).

The study also revealed significantly lower weight for age % and length/height for age % and significantly higher serum albumin in the non-edematous compared to the edematous patients (table 1).

Table (3) shows the NCV and SSEPs values in PEM patients before nutritional rehabilitation compared to that of the controls. It reveals significantly higher values of SSEPs at N₁₃, N₉₋₁₃ and N₀₋₂₀ in both groups of PEM patients compared to the controls. SSEP at N₀ was significantly lower than that of the controls in the edematous group only. Table (3) also shows that there were no statistically significant differences in NCV and SSEPs between the non-edematous and edematous PEM patients before nutritional rehabilitation.

Regarding the changes in peripheral nerve conduction velocity before nutritional rehabilitation, the current study revealed delay in ULDL in 40% of PEM patients and decrease in ULCV in 30% of them (compared to the control values). The LLDL showed delay in 40% and the LLCV was decreased in 25%. After nutritional rehabilitation there was improvement in these parameters, which decreased to 0%, 5%, 5% and 5% respectively. Figure (1) shows the results of motor nerve conduction study of a PEM patient before and after nutritional rehabilitation.

The SSEPs showed delay before nutritional rehabilitation in 25%, 55%, 30%, 100%, 10%, 55% of the PEM patients at N₀, N₁₃, N₂₀, N₉₋₁₃, N₁₃₋₂₀ and N₀₋₂₀ respectively (compared to the control values). Improvement occurred after nutritional rehabilitation and the delay was recorded at these sites only in 25%, 50%, 0%, 85%, 0% and 0% of the patients respectively. Figure (2) demonstrates the results of SSEP of a PEM patient before and after nutritional rehabilitation.

Regarding the correlation studies, there were no significant correlations between the studied NCV and SSEP values in all studied PEM patients and any of the studied anthropometric or laboratory parameters.
However, there was a negative correlation between the level of vitamin B_{12} before nutritional rehabilitation and the SSEP at N_9 (r value is –0.13) and also negative correlations between the rate of change of vitamin B_{12} and those of the SSEP at N_{13} and N_{20} (r values are –0.40 and –0.13 respectively) but as previously mentioned, these correlations were not of statistical significance.

### Table 1. Comparison between anthropometric measurements, and laboratory parameters of protein energy malnutrition patients and that of the controls before nutritional rehabilitation.

<table>
<thead>
<tr>
<th>Studied parameter</th>
<th>Group</th>
<th>Mean±SD [Median (interquartile range)]</th>
<th>Group</th>
<th>Mean±SD [Median (interquartile range)]</th>
<th>t/z* (p)</th>
<th>t/z* (p)</th>
<th>t/z* (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Non-edematous Group I n=10</td>
<td></td>
<td>Edematous Group II n=10</td>
<td>Control Group III n=10</td>
<td>Group I Vs III</td>
<td>Group II Vs III</td>
</tr>
<tr>
<td>Weight % from mean for age</td>
<td></td>
<td>51.50 ± 4.70 [52.00(4.70)]</td>
<td>61.81 ± 6.56 [61.60(8.48)]</td>
<td>94.42 ± 7.21 [91.85 (11.60)]</td>
<td>-15.30 (p&lt;0.001)</td>
<td>-10.58 (p&lt;0.001)</td>
<td>-3.90 (p&lt;0.01)</td>
</tr>
<tr>
<td>Length/height % from mean for age</td>
<td></td>
<td>87.22 ± 4.55 [86.30(6.10)]</td>
<td>91.31 ± 2.82 [90.95(4.00)]</td>
<td>96.96 ± 2.35 [96.65 (3.57)]</td>
<td>-6.01 (p&lt;0.001)</td>
<td>-4.87 (p&lt;0.001)</td>
<td>-2.42 (p&lt;0.05)</td>
</tr>
<tr>
<td>Skull circumference (cm)*</td>
<td></td>
<td>42.55 ± 2.92 [42.25(4.00)]</td>
<td>41.50 ± 1.51 [41.50(3.00)]</td>
<td>46.00 ± 2.40 [45.50 (3.00)]</td>
<td>-2.42 (p&lt;0.05)*</td>
<td>-3.67 (p&lt;0.001)</td>
<td>1.01 (p&lt;0.05)</td>
</tr>
<tr>
<td>Mid arm circumference (cm)*</td>
<td></td>
<td>8.60 ± 0.97 [8.50(1.50)]</td>
<td>9.45 ± 1.69 [9.25(3.00)]</td>
<td>12.45 ± 1.54 [13.00 (1.50)]</td>
<td>-3.52 (p&lt;0.001)*</td>
<td>-3.14 (p&lt;0.01)</td>
<td>-1.38 (p&lt;0.05)</td>
</tr>
<tr>
<td>Serum albumin (gm/dL)</td>
<td></td>
<td>4.14 ± 0.22 [4.13(0.25)]</td>
<td>1.81 ± 0.52 [1.75(0.90)]</td>
<td>4.19 ± 0.30 [4.10 (0.55)]</td>
<td>-0.23 (p&lt;0.05)*</td>
<td>-3.79 (p&lt;0.001)*</td>
<td>-3.78 (p&lt;0.001)*</td>
</tr>
<tr>
<td>Haemoglobin (gm/dL)</td>
<td></td>
<td>8.93 ± 1.00 [9.00(0.90)]</td>
<td>9.65 ± 1.78 [9.25(3.00)]</td>
<td>12.11 ± 1.63 [12.00 (2.96)]</td>
<td>-5.27 (p&lt;0.001)</td>
<td>-3.23 (p&lt;0.01)</td>
<td>-1.11 (p&lt;0.05)</td>
</tr>
<tr>
<td>Vitamin B_{12} (pgm/ml)</td>
<td></td>
<td>240.40 ± 87.98 [236.80 (139.08)]</td>
<td>227.93 ± 95.31 [261.20 (114.88)]</td>
<td>968.62 ± 177.89 [1018.55 (219.50)]</td>
<td>-11.61 (p&lt;0.001)</td>
<td>-11.60 (p&lt;0.001)</td>
<td>0.30 (p&lt;0.05)</td>
</tr>
</tbody>
</table>

*Non-parametric data detected by Shapiro-Wilk test. The test of significance used here is Mann-Whitney test. P<0.05 is significant, p<0.01 is highly significant, p<0.001 is very highly significant and p>0.05 is non-significant.

N.B.: Vs means versus
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### Table 2. Comparison between anthropometric measurements, and laboratory parameters of protein energy malnutrition patients before and after nutritional rehabilitation.

<table>
<thead>
<tr>
<th>Group</th>
<th>Studied Parameter</th>
<th>Non-edematous (n=10)</th>
<th>Edematous (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean±SD [Median (interquartile range)]</td>
<td>Mean±SD [Median (interquartile range)]</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
<td>t/z(p)</td>
</tr>
<tr>
<td>Weight % from mean age</td>
<td>51.50 ± 4.70 (52.00:4.70)</td>
<td>71.36 ± 3.90 (71.35:6.30)</td>
<td>-27.48 (p&lt;0.001)</td>
</tr>
<tr>
<td>Length/height % from mean for age</td>
<td>87.22 ± 4.55 (86.30:6.10)</td>
<td>88.74 ± 4.27 (88.80:6.10)</td>
<td>-12.64 (p&lt;0.001)</td>
</tr>
<tr>
<td>Skull circumference (cm)</td>
<td>42.55 ± 3.92 (42.25±4.00)</td>
<td>43.50 ± 3.03 (43.25±4.00)</td>
<td>-19.00 (p&lt;0.001)</td>
</tr>
<tr>
<td>Mid arm circumference (cm)</td>
<td>8.60 ± 0.97 (8.50±1.05)</td>
<td>9.55 ± 1.07 (9.50±1.05)</td>
<td>-19.00 (p&lt;0.001)</td>
</tr>
<tr>
<td>Serum albumin (gm/dL)</td>
<td>4.14 ± 0.22 (4.13±0.23)</td>
<td>4.33 ± 0.15 (4.30±0.23)</td>
<td>-2.67 (p&lt;0.01)*</td>
</tr>
<tr>
<td>Hemoglobin (gm/dL)</td>
<td>8.93 ± 1.00 (9.00±0.90)</td>
<td>9.56 ± 0.75 (9.50±1.13)</td>
<td>-7.98 (p&lt;0.001)</td>
</tr>
<tr>
<td>Vitamin B12 (pgm/ml)</td>
<td>240.40 ± 87.98 (236.80±139.06)</td>
<td>444.03 ± 106.99 (498.60±130.79)</td>
<td>2.85 (p&lt;0.01)*</td>
</tr>
</tbody>
</table>

*p<0.01 is highly significant and p<0.001 is very highly significant.

### Table 3. Nerve conduction velocity and somatosensory evoked potential values in protein energy malnutrition patients before nutritional rehabilitation compared to that of the controls.

<table>
<thead>
<tr>
<th>Group</th>
<th>Studied parameter</th>
<th>Non-edematous Group I(n=10)</th>
<th>Edematous Group II(n=10)</th>
<th>Control Group III(n=10)</th>
<th>Group I Vs III t/z*(p)</th>
<th>Group II Vs III t/z*(p)</th>
<th>Group I Vs II t/z*(p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>UL distal latency**</td>
<td>2.61 ± 0.78 (2.55:1.30)</td>
<td>2.34 ± 0.87 (2.20:1.50)</td>
<td>2.87 ± 0.19 (2.93:0.90)</td>
<td>-1.99 (p&gt;0.05)</td>
<td>-1.41 (p&gt;0.05)</td>
<td>-0.21 (p&gt;0.05)</td>
<td></td>
</tr>
<tr>
<td>UL conduction velocity ***</td>
<td>42.75 ± 6.43 (43.56:9.65)</td>
<td>43.54 ± 9.37 (45.33:13.65)</td>
<td>48.82 ± 7.23 (50.65:9.70)</td>
<td>-2.67 (p&lt;0.01)*</td>
<td>-3.40 (p&lt;0.001)</td>
<td>-1.70*p (p&gt;0.05)</td>
<td></td>
</tr>
<tr>
<td>LL distal latency**</td>
<td>2.64 ± 0.64 (2.65:1.20)</td>
<td>2.60 ± 0.55 (2.70:1.10)</td>
<td>2.71 ± 0.24 (2.82:0.36)</td>
<td>0.00 (p&gt;0.05)</td>
<td>0.46 (p&gt;0.05)</td>
<td>1.63 (p&gt;0.05)</td>
<td></td>
</tr>
<tr>
<td>LL conduction velocity ***</td>
<td>44.42 ± 8.69 (44.43:12.40)</td>
<td>50.96 ± 9.21 (50.85:4.80)</td>
<td>49.29 ± 6.69 (51.10:0.09)</td>
<td>-1.40 (p&gt;0.05)</td>
<td>0.46 (p&gt;0.05)</td>
<td>1.63 (p&gt;0.05)</td>
<td></td>
</tr>
</tbody>
</table>

*p<0.01 is highly significant and p<0.001 is very highly significant.

**Non-parametric data detected by Shapiro-Wilk test. The test of significance used here is Wilcoxon matched pairs test.

**In milliseconds, ***In meters/second

P<0.05 is significant, p<0.01 is highly significant, p<0.001 is very highly significant and p>0.05 is non-significant.

N.B: Vs means versus
Motor Nerve Conduction

(a) Delayed distal latency and decreased conduction velocity before nutritional rehabilitation.

Motor Nerve Conduction

(b) Improvement of distal latency and increased motor conduction velocity after nutritional rehabilitation.

Fig. (I): Results of motor nerve conduction study of a PEM patient
(a) before and (b) after nutritional rehabilitation.
SEP Upper Extremity

(a) Delayed SSEP at N20, N13, N9, N913 and N9-20 before nutritional rehabilitation.

SEP Upper Extremity

(b) SSEP improved latencies after nutritional rehabilitation.

Fig. (2): Results of SSEP of a PEM patient (a) before and (b) after nutritional rehabilitation.
Fig. (3): Upper and lower limb conduction velocities before and after nutritional rehabilitation in non-edematous and edematous groups.

Fig. (4): Upper and lower limb distal latencies before and after nutritional rehabilitation in non-edematous and edematous groups.
Fig. (5): Comparison between central nerve conduction latencies before and after nutritional rehabilitation in non-edematous PEM group.

Fig. (6): Comparison between central nerve conduction latencies before and after nutritional rehabilitation in edematous PEM group.
DISCUSSION

In the present work found that the central conduction time (SSEP) was more significantly affected than peripheral conduction functions in PEM patients disregards its type. The proximal central conduction time at N13-20 in our studied PEM patients did not significantly differ from the controls, which comes in agreement with a previous study done by Hesse et al. However, in the present study, we found significant delay in the distal central nerve conduction at N13, N9-13 and N9-20 denoting that the delay in central nerve conduction started distal in the pathway and began in the area between Erb’s point and the lower cervical area, which was not studied in the previously mentioned study. Fortunately, these abnormalities improved significantly after nutritional rehabilitation. Hesse and his associates studied the central nerve conduction time in a growth stunted group of children whose age range was 7-8 years and concluded that somatosensory tracts may escape myelination damage from postnatal dietary deficiencies, as it is almost complete at birth. The present study contradicts this statement and we suggest that severe malnutrition early in life (first 18 months) could have a tremendous effect on the myelination of the somatosensory tracts with subsequent neurologic sequel if not early and properly treated.

The present study found impaired myelination (presented as delayed conduction velocities) in both upper and lower limbs of the two studied groups of PEM patients compared to the controls yet this impairment was not of statistical significance. Kumar et al., reported that PEM, when it occurs during the development of the nervous system, affects myelination of the peripheral nerves. This effect of malnutrition on conduction velocities is supported by the findings of a previous study carried on 12 kwashiorkor children between the ages of one to four years. They reported delayed nerve conduction velocities in their studied patients, which was not the case when they studied children over 4 years of age. Also earlier studies on adults reported that 14-28 days starvation did not reduce the conduction velocities in peripheral nerves. Thus it is obvious that early malnutrition affects peripheral nerve conduction as well though not to the same degree as the central nerve conduction affection.

Children with protein energy malnutrition often have associated vitamin deficiencies and electrolyte defects. Before nutritional rehabilitation of PEM patients in the current study, serum vitamin B12 values were significantly lower in both studied groups compared to those of the controls. This is contrary a previous report where the authors demonstrated high vitamin B12 levels especially in edematous types of PEM. The theories they suggested were the decreased vitamin B12 requirements for erythropoiesis but this is rather improbable as the erythropoietic activity, though highly variable, is, on the average, normal in PEM patients. Also the theory of abnormal transfer of vitamin B12 from the plasma to tissues is not favored by the in-vitro studies of Grassmann and Retief. The only probable mechanism they suggested was the liver steatosis occurring in kwashiorkor causing increased vitamin B12 as observed in other causes of liver damage, yet this would not explain the same finding observed in non-edematous PEM patients.

Vitamin B12 deficiency was reported in the literature to be infrequent in PEM and respond to small doses of the vitamin. In agreement with the current study, MacDougall and Ross, Adams & Scragg, and Sandozai et al., also reported vitamin B12 to occur in kwashiorkor but infrequently.

In a study assessing the central sensory and peripheral conduction in patients with vitamin B12 deficiency, abnormal central sensory (abnormal N13 and N9-13 interval with preserved N13-20 interpeak interval), with normal peripheral conduction were reported which is in consistent with the findings of the present study. The above-mentioned abnormalities in both studies come in agreement with the pathological data of patients with vitamin B12 deficiency.
who have neurological manifestations primarily due to spinal cord lesion and that degenerative changes in peripheral nerves occur only in such patients if there is advanced forms of myelopathy\textsuperscript{31}. When we considered the whole group of PEM patients, the negative correlation detected between the level of vitamin B\textsubscript{12} before nutritional rehabilitation and the SSEP at N\textsubscript{9} and also negative correlations between the rate of change of vitamin B\textsubscript{12} and those of the SSEP at N\textsubscript{13} and N\textsubscript{20}, though not of statistical significance, point out to the fact that vitamin B\textsubscript{12} deficiency can be considered a determinant factor in the delayed central nerve conduction in the current study. The absence of statistical significance may be due to the fact that vitamin B\textsubscript{12} is only one among many neurotropic factors responsible for neural integrity.

The current study found similar results in the central sensory conduction in non-edematous and edematous groups of the PEM patients, what is considered an additional finding in our results. This was supported by the fact that no significant correlations were found between the weight \% for age or the serum albumin (which were significantly different between the two groups) and any of the neurophysiologic battery of investigations.

To explain the detected central sensory delay, we will have to refer to the critical periods of development of the tracts involved. All experts in this area agree that myelination of the different tracts takes place at different periods of time and at different rates of development, for example, the medial longitudinal fasciculus and the cuneate fasciculus show early myelination and rapid myelinative phases, myelination of the solitary tract and cervical corticospinal tract occur slow from birth to second postnatal year\textsuperscript{5}. Corticospinal tract fibers may have been at risk in our sample of patients, although we did not include corticospinal tracts in our battery of electrophysiological tests, as a previous study showed that early malnutrition in rats decreased conduction velocity of corticospinal fibers\textsuperscript{32}. Significant statistical improvement of central sensory conduction time and interpeak intervals were found in both groups of our patients after nutritional restoration. This is completely different from what was found in a previous study about early malnutrition in rats followed by nutritional restoration and reassessment 2 months later, which found persistence of the corticospinal fibers abnormality in spite of nutritional restoration\textsuperscript{32}. This supports the idea of the critical periods of development and the effect of malnutrition on CNS maturation. The significant improvement of our studied electrophysiological parameters indicates that our malnourished children escaped this critical period and nutritional restoration had a positive effect on CNS recovery and functions.

It is worth noting here that the residual delay in peripheral and central nerve conduction velocities revealed in the current study may be attributed to the short period of follow up of patients after nutritional rehabilitation and the significant improvement that occurred during that period is a good sign that longer periods of follow up may prove complete recovery of these parameters.

In conclusion, PEM disregards its type affects cortical more than peripheral myelination and this abnormality is reversible with early and optimum nutritional rehabilitation. Vitamin B\textsubscript{12} is partially responsible for this delay and the improvement of its level on nutritional rehabilitation couples that of the abnormalities in nerve conduction. We thus recommend early detection and proper management of cases of malnutrition among infants with long periods of follow up and special emphasis on providing the necessary neurotropic elements as this age has proved to be very vulnerable to neurological sequelae resulting from such deficiencies. Also, further studies are recommended to assess these patients after longer periods of follow up and study the role of other nutritional elements involved in the CNS maturation in PEM.
REFERENCES


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الملخص العربي

العلاقة بين التغييرات في التوصيل العصبي الصرفي والطرفي ومستوى فيتامين B₁₂ في حالات سوء التغذية

تحدث العديد من التغييرات في توصيل العصب الصرفي والطرفي والمكونات نتيجة نقص عدد من المغذيات الكبرى والصغير. أجريت هذه الدراسة على 30 طفلًا، عشرون منهم يعانون من سوء التغذية وعشرة من الأطفال الأصحاء كمجموعة مفيدة. وتم تقسيم المرضى إلى مجموعتين، الأولى شملت عشرة أطفال لديهم أديميا والعثرة الأخرى لم يعانون أديمية مع سوء التغذية. تم قياس نسبة فيتامين B₁₂ إلى جانب القياسات الجسدية لكل الحالات المشاركة في البحث، كما تم قياس توصيل العصب الصرفي في الأطراف السفلية وال العليا لهم في جلود أفراد المجموعة المستقرة للمسار الصرفي في أكثر من نقطة منها نقطة إرب ونقطة جزء المخ ونقطة القشرة الدماغية، كما قيست في المسافات بين هذه النقاط. تم إعادة كل هذه القياسات على حالات البحث بعد حوالي شهر من التأهيل الغذائي. أثبتت هذه الدراسة تأثير مختلف في توصيل العصب الصرفي ال�이 جزء المخ، وفي كل من نقاط أرب ونقطة جزء المخ، بالمقارنة مع المجموعة الضابطة. وكان هذا التأثير مصحوبًا بنقص ملحوظ في نسبة فيتامين B₁₂ في مجموعتي سوء التغذية على النساو مقايرًا بالمجموعة الضابطة. وقد تم التأكد من التأثير الذي يمكن أن يؤدي هذا نقص فيتامين B₁₂ في هذا المرض، وقد تحسنت هذه النتائج بعد التأهيل الغذائي، وهذا توصيف بالإسراع في تطوير برنامج التأهيل الغذائي المناسب للمرضى سوء التغذية مع التركيز على تعويض النقص في المغذيات التي تؤثر على حيوية الأعصاب وعلاقتها في فيتامين B₁₂ وذلك لتجنب المخاطر التي قد يتعرض لها الجهاز العصبي لهذه الأطفال.