Assessment of Peripheral Neuronal Activity with Nerve Conduction Studies in Iron Deficiency Anaemia Patients from Rural Areas of Bankura District of West Bengal

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Abstract
Background: People suffering from iron deficiency anaemia often complain of tingling and weakness in their limbs. In this study, we tried to explore whether there is any impairment of neuronal transmission in peripheral nerves in iron deficiency anaemia.

Materials & Methods: About 30 iron deficiency anaemia cases and 30 age sex matched non-anaemic controls were examined. All the subjects underwent nerve conduction studies. Distal latency, amplitude of compound motor action potential (cmap) and conduction velocity of bilateral median and ulnar nerves were measured. Haematological estimations were done for haemoglobin percentage, MCV, MCH and MCHC. Findings were compared between groups by unpaired students’ t-test. Association among blood parameters and NCS findings were tested by Pearson's correlation coefficient (r value). Each time p< 0.05 was considered significant.

Results: Anaemic patients showed significantly higher distal latencies but lower conduction velocity and amplitude of both median and ulnar nerves than non-anaemic controls (p<0.05 in each case). Haemoglobin percentage, MCV, MCH, MCHC showed negative correlation with latencies of median and ulnar nerves and a positive correlation with amplitude and conduction velocity (p<0.05 in each case).

Conclusion: Iron deficiency anaemia may cause impairment of peripheral nerve conduction and severity is directly proportional to haemoglobin percentage.

Keywords: Iron deficiency anaemia, Nerve Conduction studies, Distal latencies.

Introduction
It is often seen that the patients suffering from iron deficiency anaemia complain of peripheral weakness and tingling sensation in their limbs. There are many studies depicting the adverse effects of Vit B12 deficiency anaemia on nervous system.
system. But, the roles of iron deficiency anaemia in causing nerve conduction abnormalities are not well explored. It is known that iron being a part of hemoglobin carries nutrients and helps in gaseous exchange of all tissues including nerves. It also plays a role in myelin formation. But whether iron deficiency leads to nerve damage itself, is still not well documented. In this study, we tried to explore the effect of iron deficiency anaemia on nerves with the help of nerve conduction study and compare the findings with age and sex matched healthy individuals.

Materials & Methods
It was a hospital based cross sectional study. Total 60 females were examined of which 30 had iron deficiency anaemia and 30 were age and sex matched non-anaemic healthy controls. All subjects were selected from people attending the general OPD of Bankura Sammilani Medical College. The study was carried out after receiving ethical clearance from Institutional Ethics Committee and written informed consent from each subject.

Inclusion criteria: Females of 30-49years age group having iron deficiency anaemia were taken as cases and age matched non anaemic females with no other systemic illness were taken as controls. Haemoglobin< 11gms/dl, PCV< 35%, MCH<27pg, MCV < 80fl, MCHC <31 gms/dl were taken as yardsticks of iron deficiency anaemia.

Exclusion criteria: Subjects having systemic illness or any known cause of central or peripheral neuropathy, myopathy, alcoholism, pregnancy and treatment history with neurotoxic drugs were excluded. Subjects receiving iron supplementation were excluded as well.

Study design: Willing candidates were interrogated for detailed history of past and present illness as well as treatment. Complete blood count was done by fully automated haematology analyser, Analyser-PE-6800, by Aspen Diagnostics Pvt Ltd. Subjects were selected on the basis of history and test reports. All the cases having iron deficiency anaemia and the age and sex matched controls underwent nerve conduction studies in RMS EMG EP MARK II machine. Latency, amplitude and conduction velocity of compound motor action potential (CMAP) of median and ulnar nerves of both hands were taken into consideration.

Findings were compared between cases and controls.

Statistical Method
Values of individual parameters were expressed as mean and one standard deviation and were compared in groups by unpaired Student’s t-test. P<0.05 was considered as significant.

Results
Total 60 participants were assessed, among them 30 were having iron deficiency anaemia and 30 were age and sex matched non anaemic controls. Values of haematological parameters and nerve conduction parameters along with P-values are summarized below.

Table 1: Values of different study parameters

<table>
<thead>
<tr>
<th>Parameters studied</th>
<th>Anemic cases</th>
<th>Non-anaemic controls</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ulnar distal latency</td>
<td>2.72±0.48</td>
<td>1.69±0.06</td>
<td>0.015*</td>
</tr>
<tr>
<td>Median distal latency</td>
<td>2.72±0.43</td>
<td>1.62±0.32</td>
<td>0.01*</td>
</tr>
<tr>
<td>Amplitude of CMAP of ulnar nerve</td>
<td>10.40±1.27</td>
<td>14.97±1.13</td>
<td>0.023*</td>
</tr>
<tr>
<td>Amplitude of CMAP of median nerve</td>
<td>10.97±2.76</td>
<td>16.89±1.06</td>
<td>0.019*</td>
</tr>
<tr>
<td>Ulnar conduction velocity</td>
<td>53.42±9.78</td>
<td>60.16±0.64</td>
<td>0.003*</td>
</tr>
<tr>
<td>Median conduction velocity</td>
<td>58.71±5.21</td>
<td>66.08±3.40</td>
<td>0.004*</td>
</tr>
<tr>
<td>Haemoglobin conc.</td>
<td>10.16±0.34</td>
<td>11.5±1.13</td>
<td>0.012*</td>
</tr>
<tr>
<td>PCV</td>
<td>31.4±1.23</td>
<td>36.1±0.91</td>
<td>0.033*</td>
</tr>
<tr>
<td>MCV</td>
<td>73.71±0.66</td>
<td>85.85±1.29</td>
<td>0.045*</td>
</tr>
<tr>
<td>MCH</td>
<td>24.88±1.95</td>
<td>28.19±0.59</td>
<td>0.029*</td>
</tr>
<tr>
<td>MCHC</td>
<td>33.76±1.7</td>
<td>32.78±0.87</td>
<td>0.19</td>
</tr>
</tbody>
</table>

P<0.05 is statistically significant.
Distal latencies, amplitudes and nerve conduction velocities of both median and ulnar nerves were significantly different between cases and controls (P<0.05 in each case). Anaemic cases showed increased distal latencies and decreased amplitudes and conduction velocities when compared to non-anaemic controls. As for correlation coefficient, there exists significant positive correlation between haemoglobin level, PCV, MCV, and MCH with median and ulnar nerve conduction velocity and amplitude. There also exists a negative correlation between values of distal latencies with that of haemoglobin, PCV, MCV, MCH. MCHC did not show any significant correlation.

**Discussion**

In our study we found that iron deficiency alters neurological parameters. By the criterias set by WHO iron deficiency anaemia was diagnosed with the help of haemoglobin, PCV, MCV, MCH and MCHC. The probable cause of neuronal abnormalities may be that iron plays a pivotal role in structural and functional integrity of cells. It helps to carry oxygen to tissues. Being an integral part of catalases and oxidases it helps in detoxification of free radicals. It plays a role in myelination. [5, 11, 13] It helps to carry oxygen to tissues. Being an integral part of catalases and oxidases it helps in detoxification of free radicals. It plays a role in myelination. [14, 15, 16, 17]

In our case, due to defective myelination, the distal latencies have increased with decreased conduction velocity. The axon is also damaged as evidenced by decreased values of amplitude. Our findings corroborate with the findings of Y Degimenci et al. conducted on newly diagnosed anaemic patients. [18, 19, 20]

**Conclusion**

Iron deficiency anaemia leads to impaired neuronal activity and its extent is proportional to the severity of anaemia. So, iron therapy should be started at a very early stage to prevent permanent nerve damage.

**References**


