Original article:

**Nerve conduction studies in recently diagnosed untreated hypothyroid patients.**

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**Abstract**

Objectives- The purpose of this study is to evaluate objectively the functional changes in peripheral nerves of recently diagnosed untreated hypothyroid subjects by electrodiagnostic tests in order to estimate the frequency of nerve conduction abnormalities.

Methods- A cross sectional analytical study was performed in adult patients recently diagnosed with thyroid dysfunction, but not placed on hormone replacement therapy. Patients with biochemical evidence of hypothyroidism were enrolled for the study. Nerve conduction study was carried out and parameters namely distal latency, amplitude and conduction velocity were assessed in median, ulnar, peroneal and sural nerves.

Results- 75% patients had distally located paresthesias, 55% complained of cramps and 15% had weakness of the lower limbs. In 19% subjects both motor and sensory nerves were involved, while in 69% there was involvement of only sensory nerves. There was slowing of nerve conduction velocity in 71% patients characteristically for median and sural sensory nerves. Carpal tunnel syndrome was seen in 15% patients.

Conclusion –Neuromuscular symptoms were commonly encountered. There was a predominant involvement of sensory nerves, especially the sural nerve and median nerve on nerve conduction study. Early detection can prevent structural alterations in peripheral nerves as they occur later in the course of the disease and help in initiating replacement therapy at the earliest.

Keywords: Nerve conduction, Hypothyroidism, Polyneuropathy

**Introduction**

Thyroid hormones have a multitude of systemic effects. Hypothyroidism is a chronic disease affecting a wide variety of systems such as excretory, digestive, cardiac and nervous [1,2]. Peripheral nervous system involvement in hypothyroidism is a well documented fact [3,4,5,6]. The reported prevalence of neuromuscular disorders related to thyroid dysfunction is 20-80% [7, 8] , the severity of which correlates with the degree and duration of hormonal deficiency. In fact neurological complications in hypothyroidism are a well established finding. Peripheral nerve abnormalities known to be associated with hypothyroidism are entrapment neuropathy or sensorimotor polyneuropathy. Retrospective electrophysiologic studies in patients with thyroid disease undergoing treatment are reported [6, 9]. Hence, the objective of the current study was a) To conduct nerve conduction studies in patients with recently diagnosed untreated hypothyroidism .b) To assess the degree of impairment of peripheral nerve function c) To compare the latency, amplitude , conduction velocity of some nerves in patients with that of control .d) To calculate the frequency of nerve conduction abnormalities in these patients.

**Material and methods**

The case control study was conducted at Government Medical College, Akola (MS) between September 2012 and October 2013. Adult patients attending the outpatient department who were advised thyroid function tests were isolated and only those patients with biochemical evidence of hypothyroidism i.e. low Tri –iodothyronine and Thyroxine levels with Thyroid stimulating hormone levels were enrolled for the study. These were recently diagnosed cases who were not on any treatment for hypothyroidism. Patients with diabetes mellitus, neuromuscular disorders limb injury, neuromuscular transmission disorder, myopathy or with a history of neuropathy were excluded. Similarly alcoholics, smokers, subjects on medication affecting central or peripheral nervous system were excluded. History
pertaining to principal symptoms of thyroid disease was obtained, this included alopecia, weight gain, cramps, weakness, myalgia, tremors and intolerance to cold. All participants were examined to rule out any systemic, neuromuscular, cognitive dysfunction or psychiatric disorder. Hypothyroid subjects were asked about their neuromuscular symptoms and whether it was their first complaint. Relevant clinical history was taken and clinical examination done in all subjects. Age and gender matched healthy subjects with normal thyroid function tests were chosen as the control group for comparison. Approval of institutional ethical committee was taken. Written informed consent was obtained from all participants.

Nerve conduction study was done using RMS EMG EP Mark2. The Motor nerves tested were median, ulnar and peroneal. The Sensory nerves studied were median, ulnar and sural. The parameters which were studied were distal motor latency, amplitude and conduction velocity for motor nerves. Distal latency, Amplitude, conduction velocity and antidromic study was carried out for sensory nerves. Nerve conduction studies were conducted in hypothyroid patients as well as the control group. Data was collected by the same examiner, between 11 am to 1pm. Mean values with standard deviation of the said parameters were calculated. SPSS Software 10.0 version was used for statistical analysis. 'p' value was considered statistically significant if found to be less than 0.05.

Results
Mean age of the participants was 35.5 years. Age groups were not significantly different between cases and controls. (Table 1) Of the patients 22 were females and 4 males. Neuromuscular complaints for the hypothyroid group were fatigue, myalgias, cramps and paresthesias. Neuromuscular complaints as primary complaints were seen in 16 [69%] patients. Paresthesias were the most common complaint 75%, followed by cramps 55% and weakness 15%. Electrophysiologic studies were conducted in twenty six patients and thirty subjects of the control group. On evaluation of the results, in comparison to the normal values obtained from the controls, 23 [88%] had abnormal nerve conduction studies. Both motor and sensory nerves were affected in 5 (19%) whereas Sensory nerves alone were affected in 18 (69%) patients (Table 2). Table 3 shows the comparison between hypothyroids and controls for the mean values of parameters of nerve conduction studies. Polyneuropathy was labelled when two values were found abnormal on nerve conduction studies in at least two nerves. Prolonged distal latencies and reduction in amplitude of compound muscle action potential on proximal stimulation of nerve as compared to distal stimulation with slowing of motor conduction velocities was observed in median and peroneal motor nerves of 10% patients but the difference was not statistically different from the control group. Antidromic study revealed prolonged distal latencies and reduced amplitude of sensory nerve action potential with slowing of conduction velocity in sural and median sensory nerves of 18(69%) patients. This reduction was statistically significant from the control group. Entrapment neuropathy was observed in 4 [15%] patients while sensory polyneuropathy in 15 (57%) patients. The parameters were not recordable in three subjects.

### Table 1: Age and Gender distribution

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Hypothyroid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in yrs</td>
<td>34.5± 1.4*</td>
<td>37.4± 2.1*</td>
</tr>
<tr>
<td>Males</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Females</td>
<td>27</td>
<td>22</td>
</tr>
</tbody>
</table>

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TABLE 2: Involvement of nerves

<table>
<thead>
<tr>
<th>Nerves</th>
<th>No. Involved.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory</td>
<td>18</td>
<td>69</td>
</tr>
<tr>
<td>Both motor and sensory</td>
<td>5</td>
<td>19</td>
</tr>
</tbody>
</table>

TABLE 3: Comparision of Nerve conduction parameters (values in mean ± SD)

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Conduction velocity (m/s)</th>
<th>Latency (s)</th>
<th>Amplitude (mv)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Hypothyroid</td>
<td>Control</td>
</tr>
<tr>
<td>Median motor</td>
<td>58.3± 3.3</td>
<td>56.2± 2.9*</td>
<td>3. ± 0.3</td>
</tr>
<tr>
<td>Median sensory</td>
<td>56.9± 4.2</td>
<td>52.7± 3.8**</td>
<td>3.1± 0.5</td>
</tr>
<tr>
<td>Ulnar motor</td>
<td>59.7± 4.1</td>
<td>59.3± 3.1*</td>
<td>2.6± 0.4</td>
</tr>
<tr>
<td>Ulnar sensory</td>
<td>57.6± 4.4</td>
<td>56.7± 3.8*</td>
<td>2.9± 0.5</td>
</tr>
<tr>
<td>Peroneal motor</td>
<td>52.2± 2.6</td>
<td>51± 3.2*</td>
<td>4.1± 0.5</td>
</tr>
<tr>
<td>Sural sensory</td>
<td>57.6 ± 4.1</td>
<td>52.4 ±4.1**</td>
<td>3.3 ±0.3**</td>
</tr>
</tbody>
</table>

* - Statistically not significant  ** -- Statistically significant

Discussion

Peripheral nervous system dysfunction is an important outcome of thyroid hormone deficiency. In newly diagnosed cases symptoms and signs of mixed neuropathy may predominate initially. The symptoms of peripheral nerve involvement are a fairly sensitive predictor of polyneuropathy. [6]Majority of these cases would have gone unnoticed, had only a clinical examination been done. Often polyneuropathy in hypothyroidism is mild and rarely it could be a subclinical entity[ 6]. But clinical examination alone may be insufficient to make a conclusive diagnosis especially to make a distinction between axonal and demyelinating disorders . At such times electro diagnostic analysis is particularly useful. They assess the functional integrity of sensorimotor conduction and give a reliable evidence of peripheral nerve dysfunction. In fact the estimated prevalence of polynueopathy diagnosed by electro physiologic tests is 718/1000 [6].

The commonly reported subjective complaints of paresthesias and pain in extremities were encountered in our study too. Paresthesias were found in 75%, it correlates with other studies wherein it was 64% and 68%.[6,10].Cramps 55%and weakness 15%were the next predominant symptoms. On neurological examination slowing of reflexes was noted in only 2% cases . In our study 88% of hypothyroid patients had at least one type of electrophysiological abnormality, most commonly in median and sural sensory nerves. Sural and median nerves are
involved earlier in our study; conduction velocity was abnormal in 69% cases, and in median nerve in 13% cases by Ettore B. This could be due to a distoproximal progression of polineuropathy as reported in earlier studies. [9,11] Although motor nerve conduction velocity could be within a normal range sensory nerve action potentials are reduced at an early phase of the disease [12]. Reduction in amplitude in 60% cases for median and sural sensory nerves and slowing of conduction velocity in 71% for these nerves was noted. Rao et al[13] found reduction of amplitude for median sensory nerves whereas Fincham[14] found it for median and ulnar sensory nerves. Our data comprising of outpatients with thyroid dysfunction confirms the assumption that demyelinating polyneuropathy in hypothyroidism is commonly encountered. The nerve conduction study findings correlate well with those in literature. Axonal degeneration has been reported both electrophysiologically and pathologically [9]. Previous studies have shown a reduction in amplitude and mild slowing of sensory and motor conduction velocity consistent with presence of axonal polyneuropathy [7]. Although, such findings were not observed in our study. Morphological evidence of primary axonal degeneration with secondary demyelination has also been cited in studies [15]. Entrapment at the wrist is a part of the widespread involvement of peripheral nervous system, manifesting as carpal tunnel syndrome. We encountered CTS in five patients (19%). CTS was the second most frequent finding by Marcia Cruz, CTS symptoms being present in 71.42% of the patients who had electrodiagnostically confirmed CTS. Incidence of CTS is 5-92% [3,8] of hypothyroid patients.

Neurological dysfunction associated with disorders of thyroid gland could be the result of hormonal imbalance or immune mechanism accompanying thyroid disease [7,8,16]. Metabolic alteration in hypothyroidism affects Schwann cells leading to segmental demyelination [11] which is reflected as a decrease in conduction velocity. Since thyroid hormones are involved in gene expression, myelin production, neurotransmitter system and axonal transport, the plausible mechanism of axonal neuropathy is a hypothyroidism induced ATP deficiency with reduced activity of ATPase enzyme. This in turn induces a decrease Na-K pump activity and subsequently alteration in pump dependent axonal transport [9,17]. CTS is attributed to mucinous material in tissue around the median nerve accompanied by hypothyroid induced demyelination. [3,9] Limitations – Repetition of the electrodiagnostic study could have helped in evaluating the course of neuromuscular dysfunction in these patients. EMG was not done on these patients simultaneously; it would give a conclusive evidence of associated myopathy. Similarly somatosensory evoked potentials would be helpful and diagnostic in asymptomatic patients especially.

**Conclusion**

In hypothyroidism, metabolic alterations initially affect function and then induce structural alterations. Our study shows that hormonal and metabolic changes which are responsible for electrophysiological changes may occur early in the disease course and manifest prior to the other presentation of thyroid disease. So we suggest performing nerve conduction tests in hypothyroid patients early in the course of the disease, as a routine even in those asymptomatic for nervous complaints, to minimize structural damage and disability.

**References**