Hypothyroidism Effect on Motor Nerve Conduction Studies: A Cross Sectional Study

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Introduction

The role of thyroid hormone is vital in the timing and pace of development of central nervous system of our body during intrauterine life and in the early infancy. If there is a deficiency of thyroid hormone, it will greatly influence the growth of the cerebral and cerebellar cortex, proliferation of axons and branching of dendrites, myelination and cell migration. Deficiency of thyroid hormones during neural differentiation results in severe and permanent modifications in the morphology and function of the nervous system, in particular a general slowing of cognitive functions with memory impairment and apathy.[1]

One of the manifestations of the hypothyroidism is the peripheral neuropathy. The development of this neuropathy is insidious in onset, which will take a long period of time for clinical manifestations.[2] The severity of the neuromuscular signs and symptoms depends on duration and severity of hormonal deficiency and clinical, electrophysiological and morphological improvement with hormone replacement therapy is typical.[3-5]

Aims and Objectives

The aim of the study is to analyze the motor nerve abnormalities in hypothyroid patients and compare it with non hypothyroid controls.

Materials and Methods

After approval of the study from the ethical committee of J.L.N. Medical College; valid written and informed consent was taken from all subjects. Patients of both genders were included in the study. One hundred subjects were taken, out of which fifty were euthyroid control and fifty were hypothyroid.

Inclusion Criterion: Patients who are within 18-60 yrs age group, patients with known case of hypothyroidism, newly diagnosed as well as on medication but not attaining euthyroid state were taken.(Hypothyroid was defined as having raised serum TSH level and decreased free thyroxine (FT4) level.)

Exclusion Criterion: Patients having other causes of neuropathy like Diabetes mellitus, renal failure, Neuropathies associated with toxic agents e.g. metal or drugs, Neuropathies associated with malnutrition, alcoholic hepatitis or medication...
were excluded. Patients having skin lesion or swelling that would interfere with nerve conduction study, previous trauma to the study site were also excluded.

Method: Nerve conduction studies (NCSs) was performed on hypothyroid patients (newly diagnosed or on treatment for hypothyroidism but not attaining euthyroid state) and healthy individuals those who had given consent to do so by electrophysiological method. The NCS was performed at room temperature, with normal body temperature, on EMG NCV EP machine by Recorders and Medicare Systems, model RMS SALUS 4C. The state of hypothyroidism was detected by measuring Free T3, Free T4 and TSH by Chemiilluminisence method. Median and Ulnar nerves of the upper limbs and peroneal nerves of the lower limbs were tested. Latency, Amplitude, and Conduction velocity were noted and compared with age specific reference data of our electrophysiology laboratory under the Neurophysiology lab at JLN Hospital and Medical College, Ajmer.

The statistical analysis was done by unpaired t Test

Results

Table 1: Comparison between Motor Nerve Conduction Parameters in both the Hypothyroid (n=50) and the control subjects (n=50).

| PARAMETER | NERVE | GROUP   | NUMBER | MEAN±SD | P – Value |
|-----------|-------|---------|--------|---------|-----------|
| Latency (ms) | Rt median nerve | Controls | 50     | 7.80±0.58 | <0.001    |
|           |       | Cases   | 50     | 8.59±1.20 |           |
|           | Lt median nerve | Controls | 50     | 7.62±0.46 | <0.001    |
|           |       | Cases   | 50     | 8.59±1.19 |           |
|           | Rt ulnar nerve | Controls | 50     | 6.56±0.58 | 0.592     |
|           |       | Cases   | 50     | 6.38±2.29 |           |
|           | Lt ulnar nerve | Controls | 50     | 7.03±0.63 | 0.119     |
|           |       | Cases   | 50     | 6.55±2.05 |           |
|           | Rt COMMON PERONEAL | Controls | 50     | 10.35±0.88 | 0.117     |
|           |       | Cases   | 50     | 11.14±3.40 |           |
|           | Lt COMMON PERONEAL | Controls | 50     | 10.28±0.81 | 0.311     |
|           |       | Cases   | 50     | 10.77±3.29 |           |
| Amplitude (mV) | Rt median nerve | Controls | 50     | 9.92±1.97 | <0.001    |
|           |       | Cases   | 50     | 6.42±2.92 |           |
|           | Lt median nerve | Controls | 50     | 11.66±3.32 | <0.001    |
|           |       | Cases   | 50     | 6.40±2.29 |           |
|           | Rt ulnar nerve | Controls | 50     | 11.77±2.43 | 0.45      |
|           |       | Cases   | 50     | 11.40±2.45 |           |
|           | Lt ulnar nerve | Controls | 50     | 12.03±2.37 | 0.198     |
|           |       | Cases   | 50     | 11.40±2.49 |           |
|           | Rt COMMON PERONEAL | Controls | 50     | 4.04±1.92 | 0.06      |
|           |       | Cases   | 50     | 3.35±1.07 |           |
|           | Lt COMMON PERONEAL | Controls | 50     | 4.68±1.56 | 0.088     |
|           |       | Cases   | 50     | 4.17±1.39 |           |
| NCV (m/s) | Rt median nerve | Controls | 50     | 58.50±4.59 | <0.001    |
|           |       | Cases   | 50     | 50.38±8.53 |           |
|           | Lt median nerve | Controls | 50     | 58.66±5.02 | <0.001    |
|           |       | Cases   | 50     | 51.27±7.99 |           |
|           | Rt ulnar nerve | Controls | 50     | 59.65±7.15 | 0.877     |
|           |       | Cases   | 50     | 59.41±8.22 |           |
|           | Lt ulnar nerve | Controls | 50     | 60.12±6.96 | 0.958     |
|           |       | Cases   | 50     | 60.04±8.25 |           |
|           | Rt COMMON PERONEAL | Controls | 50     | 51.95±9.5 | 0.278     |
|           |       | Cases   | 50     | 50.15±6.76 |           |
|           | Lt COMMON PERONEAL | Controls | 50     | 51.90±4.74 | 0.269     |
|           |       | Cases   | 50     | 50.50±7.53 |           |
Mean age of cases were 45.94±12.97, and for control group were 46.1±11.5. There was no significant difference in age between cases and control group. Sex and BMI matching was also not significant.

Mean Motor Nerve Conduction Velocity of Median nerve on right side in Cases was 50.38±8.53 and that of Control group was 58.50±4.59. Mean Motor Nerve Conduction Velocity of Median nerve on left side in Cases was 51.27±7.99 and that of Control group was 58.66±5.02. A Significant decrease in MNCV of the Median nerves on both sides in hypothyroid subjects was observed as compared to the control subjects.

MNCV of Ulnar and Common peroneal nerves on both side did not show significant decrease in hypothyroids as compared to the control subjects.

Motor Latency of Median Nerve on Right side in Cases was 8.59±1.20 and in control group was 7.80±0.58. Motor Latency of Median Nerve on left side in Cases was 8.59±1.19 and in control group was 7.62±0.46.

There was significant increase in latency on both sides in hypothyroid subjects as compared to the control subjects. Motor Latency of Ulnar and Common peroneal nerves on both sides did not show significant increase in Hypothyroid, as compared to the control subjects.

Motor amplitude of Median Nerve on Right side in Cases was 6.42±2.29 and in control group was 9.92±1.97. Motor amplitude of Median Nerve on left side in Cases was 6.40±2.29 and in control group was 11.66±3.32.

A Significant decrease in motor amplitude of the Median Nerve on both sides in hypothyroid subjects was observed as compared to the control subjects.

Amplitude of Ulnar and Common peroneal nerves on both sides did not show significant decrease in hypothyroid, as compared to the control subjects.

By our nerve conduction study, it is found that 72% of hypothyroid patients have neuropathy.

On analysis of the nerve conduction study values of the three (median, ulnar, common peroneal) nerves on patients and controls the findings are follows:

a) 72% of hypothyroid patients show electrophysiological changes suggestive of neuropathy
b) 56% of the patients show sensory abnormality in the median nerve conduction, so the upper limb is more affected than the lower limb.
c) Considering the parameters most affected, the conduction velocity is the most affected parameter (72%), next is latency followed by amplitude.

**Discussion**

Thyroid hormone is known to influence the synthesis of protein and the production of enzyme and myelin sheath.\(^\text{[6,7]}\) Myelin synthesis is an important factor in determining the speed of impulse transmission along the nerve length.\(^\text{[8]}\) Disturbed myelin synthesis during acute hypothyroidism may be the cause for demyelinating peripheral neuropathy in hypothyroid patients. Hormonal and metabolic changes associated with hypothyroidism are responsible for the electrophysiological changes in the form of abnormal peripheral nerve conduction study which occurs early in the disease course.

In our study, the MNCV of median nerve was found to be significantly decreased on right as well as left side in hypothyroid subjects as compared on the MNCV of median nerves in control subjects. Also motor latency of median nerve was increased in hypothyroid subjects as compared to motor latency of control subjects and reduced CMAP amplitudes. These findings for motor conduction parameters in hypothyroids are consistent with that reported by Kececi H et al 2006,\(^\text{[9]}\) El-Salem K et al 2006\(^\text{[10]}\), Sabina et al 2008,\(^\text{[11]}\) and Somay et al.\(^\text{[12]}\)

Yüksel et al in their study also found the same results but their sample size was small.\(^\text{[13]}\) Cruz et al, (1996) found that 71.42% of cases classified as Carpal Tunnel Syndrome (CTS) by Motor Nerve Conduction Velocity (MNCV) had symptomatic and with the same incidence,
Tinel sign and median nerve territory hypoesthesia were observed.\cite{14}

Asymptomatic patients were examined by El-Salem & Ammari., (2006) \cite{10} to determine the frequency and pattern of electromyography and nerve conduction studies (NCS) changes in these patients and to see if these changes are reversible or not. Motor neuropathy was more common than sensory neuropathy affecting distal latencies more often than compound muscle action potential amplitudes. These findings favor a demyelinating rather than an axonal process. The median nerve was the most commonly affected nerve (30\% of patients). The pattern of involvement was consistent with carpal tunnel syndrome, as it showed slowing of nerve conduction across the wrist.\cite{7}

In our study 72\% of hypothyroid patients had at least one type of electrophysiological abnormality, most commonly in median nerve.

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 electro physiologically and pathologically. \cite{11}

Previous studies have shown a reduction in amplitude and mild slowing of motor conduction velocity consistent with presence of axonal polyneuropathy.\cite{15}

Morphological evidence of primary axonal degeneration with secondary demyelination has also been cited in studies. \cite{16}. Entrapment at the wrist is a part of the widespread involvement of peripheral nervous system, manifesting as carpal tunnel syndrome. We encountered CTS in none of the patients .CTS was the second most frequent finding by Marcia Cruz, CTS symptoms being present in 71.42\% of the patients who had electro diagnostically confirmed CTS Incidence.

The neurological abnormalities associated with hypothyroid patients may be a result of hormonal imbalance or may be related to the immune mechanisms related with thyroid diseases.\cite{17}

Some investigators suggested that the weight gain in the hypothyroid patients may be a contributory factor for neuropathy. Entrapment of median nerve at the wrist caused by the deposition of mucinous material in the tissues surrounding the nerve is one of the most frequent causes of peripheral nerve damage in hypothyroidism.\cite{17,18}

The thyroid hormones also increase the ATPase activity and consequently Na-K pump activity in normal individuals. Consequently, the deficiency of ATP, reduced ATPase and Na-K pump activity in hypothyroidism cause subsequent alteration of pump dependent axonal transport and may lead to peripheral neuropathy.\cite{11,18} The peripheral nerve dysfunction was also linked to the morphological evidence of primary axonal degeneration in the form of shrinkage of axons, disintegration of neurotubules and neurofilaments and active axonal breakdown.\cite{10} Some investigators studied the patients of hypothyroidism morphologically and neurophysiologically and suggested that the metabolic alterations caused by hypothyroidism were responsible for the peripheral neuropathy.\cite{17,18} However, the neuropathy due to compression and that due to axonal degeneration are not fully distinguished. Combination of both of these may result in the development of peripheral neuropathy in hypothyroidism. The motor neural dysfunction seen in the present study may be linked to the various functional and structural changes in peripheral nerves associated with deficiency of thyroid hormones.

**Conclusion**

The median nerve is the most common affected motor nerve .Estimation of the nerves conduction values can be considered as a useful parameter in the diagnosis and evaluation of the neuropathy in hypothyroid patients.

We conclude that hypothyroidism causes significant decrease in NCV and amplitude as well as increase in latency, and this decrease in NCV is more prominent in median nerve. It is proposed that as soon as a patient is diagnosed with
hypothyroidism they should be evaluated for decrease in NCV. 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.

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