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Neurological manifestations and electrophysiological findings in patients with de novo detected hypothyroidism.

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Abstract

Introduction: Hypothyroidism is a very common condition. The most common neurological symptoms caused by hypothyroidism are dementia slowed mental processing, depression, nerve entrapment syndromes, ataxia, muscle weakness and muscle cramps. Recognition of these complications early in the course of the disease is important because prompt and early treatment of hypothyroidism will reverse these complications.

Material & Methods: This prospective study included 35 de novo diagnosed hypothyroidism patients during August 2013 and November 2014. Neurological Examination included assessment of cognitive functions by Folstein Mini Mental Status Examination (MMSE) which is a 30 point questionnaire. Electrophysiological studies were performed using Medelec synergy (VIASYS Health Care, USA) at electrophysiology lab. Nerve conduction studies were done for all patients and age-matched controls in the same laboratory.

Results: Cognitive dysfunction was seen in 5.7% of our patients. Depression was noted in 14% of our patients. Muscle pains and cramps were seen in 11.4% and one patient had proximal muscle weakness in lower limbs. Nerve conduction studies revealed that 51% of patients with de novo hypothyroidism had electrophysiological evidence of neuropathy. BAEP evaluation in the present study showed that both peak latencies and interpeak latencies are prolonged, suggesting that nervous system dysfunction seen in hypothyroidism is diffuse in pattern, affecting both peripheral conduction time and central conduction time.

Conclusion: Hypothyroidism is a common condition causing neurological complications involving both central nervous system and peripheral nervous system. It is recommended that thyroid function tests should be included in the work up of dementia, depression, parasthesias and myopathies. BAEP evaluation suggesting that nervous system dysfunction seen in hypothyroidism is diffuse in pattern, affecting both peripheral conduction time and central conduction time.

Keywords: Hypothyroidism; electrophysiology; motor nerve; sensory nerve; Carpal tunnel syndrome.

Introduction

It is estimated that about 2% of adult women and 0.1-0.2% of men have clinical hypothyroidism. The prevalence of subclinical disease is more frequent, up to 9% of adult population.[1] The prevalence of hypothyroidism in Indian urban population is 10.95%.[2]

The variety of end-organ effects and wide range of disease severity from entirely asymptomatic individuals to patients in coma can make hypothyroidism an elusive clinical entity.[3] Dementia, slowed mental processing, depression, nerve entrapment syndromes, ataxia, muscle weakness and muscle cramps are the most common neurological symptoms caused by hypothyroidism.[3] Recognition of these complications early in the course of the disease is important because prompt and early treatment of hypothyroidism will reverse these complications.

Most of the studies regarding patients with thyroid diseases are retrospective and involved patients under treatment. The present study was undertaken to evaluate the neuromuscular symptoms and signs, as well as the electrophysiological changes in patients with de novo diagnosed hypothyroidism before starting treatment for the same.

Material & Methods

This prospective, observational study was conducted in the department of Neurology, Sri Venkateswara Institute of Medical Sciences (SVIMS), Tirupati. A total of 35 de novo diagnosed hypothyroidism patients were enrolled between August 2013 and November 2014.

Inclusion criteria: Patients aged between 20 and 50 years with de novo diagnosis of hypothyroidism with biochemical evidence of primary hypothyroidism (i.e., TSH > 10 µIU/ml and T4 < 55 ng/ml) with or without neurological complaints, before starting treatment were enrolled into the study.

Exclusion criteria: Patients with other comorbidities - Diabetes Mellitus, Alcoholism, Neuromuscular Disorder, Drug induced Neuropathy, Family history of Neuropathy, Malignancy, HIV, Liver diseases and Kidney Disease were excluded.

Control Group: 20 euthyroid adults aged between 20 and 50 years from hospital staff and the relatives of patients were studied as controls.

Regulatory approvals: The study was approved by Institute Ethics committee and a written informed consent was taken from each case and control.

Clinical Evaluation: Detailed history was taken pertaining to the symptoms, history of diabetes mellitus, kidney disease, malignancy, alcoholism, drug history that could cause neuropathy and family history. General clinical examination with special attention to skin for dryness, loss of hair over lateral side of eyebrows, speech for any hoarseness and local examination of neck for any swelling, its size, consistency and tenderness was done. Neurological Examination included assessment of cognitive functions by Folstein Mini Mental Status Examination (MMSE) which is a 30 point questionnaire.[4]

Laboratory Investigations: Serum TSH and T4, Hemogram, Fasting and Post Prandial blood sugars, serum creatinine were estimated.

Serum T4 was measured by chemiluminescence method on the Beckman Coulter Access immunoassay system (Beckman Coulter Ireland, Inc, Galway, Ireland). Serum TSH was measured by chemiluminescence method on the Beckman Coulter Access 2 immunoassay system (Beckman Coulter Ireland, Inc, Galway, Ireland). Plasma glucose levels were measured by glucose oxidase-peroxidase method using Beckman syncron CX5 autoanalyzer (Beckman Coulter, California, United States of America). Serum creatinine was measured with

modified Jaffe's method using Beckman system pack on Beckman syncron CX5 autoanalyzer (Beckman Coulter California, USA).

Electrophysiological studies: Electrophysiological studies were performed using Medelec synergy (VIASYS Health Care, USA) at electrophysiology lab.

Motor nerve conduction velocities (CV), distal latency and amplitude of compound muscle action potentials (CMAPs) were measured with standard surface stimulating and recording techniques for median, ulnar, common peroneal and posterior tibial nerves of both sides. Sensory nerve conduction velocities were measured orthodromically in ulnar and median nerves (5^{th} and 2^{nd} finger-wrist), and antidromically in sural nerve. The room temperature was maintained above 25^0c. The normal limits of CV and distal latency were set 2 SD from the mean values for these controls. The action potential was considered abnormal if the amplitude was below the lowest value found in the controls. An electrophysiological diagnosis of polyneuropathy was made when at least two parameters were abnormal in at least two separate nerves. The mean values for each electrophysiological parameter in hypothyroid patients were compared with standard values obtained in controls using Student's t test.

Brainstem Auditory Evoked Potentials (BAEP) were recorded for 25 patients by using headphones and the sounds used were clicks. 1000 click stimuli at the rate of 10 Hz with duration of 0.1 ms were delivered at 60 dB above hearing threshold through shielded head phones with –40 dB white noise masking the contralateral ear. Signals were filtered with bandpass 50 Hz and 3 KHz and averaged to 1000 stimuli. Active electrodes were placed on the earlobes. The reference electrode was attached to the scalp about 5 cm behind the vertex, and the ground electrode was placed at the midline of the forehead. The waves routinely analysed in BAEPs were numbered (I–V). The absolute latency (stimulus to peak) of each wave (I, II,III,IV and V), interpeak latencies (I–III, I–V and III–V) and amplitude of waves I and V were measured for each ear separately with the help of digital cursors. The amplitude was measured as the maximum height of the peak from the succeeding trough. The mean values for each wave in hypothyroid patients and age matched controls were compared using Student's t test.

Statistical Analysis

Data was recorded on a pre designed proforma and managed using Microsoft Excel 2007 (Microsoft Corp, Redmond, WA). All the entries were double checked for any possible error. Mean and standard deviation (S.D.) of demographic characteristics and all electrophysiological parameters was calculated. Comparison of means between patients and controls was done by Independent Student's t-test. Chi-square test was used to compare the proportions. P-value of $\leq$ 0.05 was considered significant. SPSS version 15.0 (SPSS Inc, Chicago, IL, USA) was used for statistical analysis.

Results

The mean age of patients (n=35) with hypothyroidism was 35.8 ± 10.1 years and was not different from that of control group (n=20, 34.4 ± 8.9 years) (p=0.6). Among the patients, the number of female participants was 27 (77%), while that of the male participants was 8 (23%). In the control group, the number of females were 14 (70%), and males were 6 (30%) (p=0.3).

Generalized weakness and easy fatiguability was the most common complaint reported by 18 patients (57%), followed by paresthesias of limbs by 10 (28.6%), muscle pains and cramps by 4 (11.4%), proximal muscle weakness in lower limbs by 1 patient and distal lower limb weakness by 1 patient. General examination showed dry skin in 4 patients (11.4%) and thyroid swelling in 3 patients (8.6%).

Neurological examination revealed low MMSE score for their respective education level in 2 patients (5.7%) in cognitive testing. Among the 2 patients 1 had difficulty in recall while the other had difficulty in recall, attention and calculation. Sensorineural type of deafness was seen in 2 patients (5.7%). Depression fulfilling the DSM IV criteria was noted in 5 patients (14.28%).

Neuromuscular examination showed muscle weakness in 2 patients (5.7%). One had weakness of distal muscles in the lower limbs in the form of bilateral foot drop with bilateral dorsiflexion of foot of grade 2/5 and plantar flexion of grade 3/5 on both sides. Other patient had proximal muscle weakness in lower limbs of grade 4 power with normal power in upper limbs and in distal muscles. Rest of the patients had normal power. Deep tendon reflex examination revealed sluggish reflexes in 5 patients (14.3%) and areflexia in 3 patients (8.6%), a total of 8 patients (22.8%).

Delayed relaxation of ankle jerk (Woltman's sign) was seen in 2 patients (5.7%). Sensory examination revealed decreased sensation of fine touch in lower limbs below the ankle and decreased sensation of vibration in lower limbs up to ankle in 2 patients (5.7%).However pain and proprioception sensation were normal in these patients. Cerebellar signs were not seen in any of the patients. Gait in the patient with foot drop was high steppage gait while the rest of the patients had normal gait.

Electrophysiological studies

Nerve Conduction study Findings: Motor nerve conductions showed decreased conduction velocities in hypothyroid patients in median (p=0.009) and ulnar nerves (p=0.03). Compound muscle action potential amplitudes and latencies were not different among patients and controls in median and ulnar nerves. The compound muscle action potential amplitudes were significantly reduced (p=0.04) in common peroneal nerve motor conductions in hypothyroid patients compared to control group, whereas latencies and conduction velocities were similar in both the groups. Posterior tibial nerve motor conductions were not different among patients and controls. Motor nerve conduction study findings are summarized in table 1.

Sensory nerve conductions showed decreased amplitudes (p=0.05) and conduction velocities (p=0.04) in median nerve in hypothyroid group, whereas latencies were similar among patients and controls. Conduction velocities were significantly decreased (p=0.04) in ulnar nerve conduction studies. Latencies and compound muscle action potential amplitudes were not different among patient and control groups in the ulnar nerve. Sural sensory nerve conductions were similar in both the groups. Sensory nerve conduction study findings are summarized in table 2.

Prevalence of Neuropathy in Hypothyroid patients: Sensory motor polyneuropathy was noted in 6 patients (17%). Sural sensory mononeuropathy was seen in 4 patients (11.43%). Isolated Carpal tunnel syndrome (CTS) was seen in 2 patients (5.71%). Motor neuropathy was seen in 4 patients (11.43%). Subclinical involvement was found in 14% of cases by ENMG. Findings are summarized in table 3.

Brainstem auditory evoked potentials (BAEP): Brainstem auditory evoked potentials (BAEP) from both the ears showed statistically significant ($p<0.05$) prolonged peak latency of wave V (5.51 ± 0.3 vs 5.34 ± 0.23, p=0.04) and I-III (1.35 ± 0.5 vs 1.12 ± 0.13, p=0.05), I-V (3.44 ± 0.42 vs 3.24 ± 0.16, p=0.05) inter peak latency, while there was no significant change in the latency of other waves and III-V interpeak latency and decrease in amplitudes of wave I and wave V was statistically not significant.

Discussion

In the present study the mean age of hypothyroid group was 35.77 ±10.1 years which was similar to the study from Iraq [5] (32.1± 6.4 years), which studied the prevalence of neuromuscular abnormalities in 58 patients of newly diagnosed hypothyroid patients over a period of two years. Another study from Egypt [6] in which patients with newly diagnosed hypothyroidism were studied for electrophysiological alterations in central and peripheral nervous system over a period of two years had a mean age of 38.2±13.6. The female preponderance for hypothyroidism in the present study (77%) was comparable to that of the study from Iraq [5] where out of 58 hypothyroid patients the number of female participants was 42 (72.4%). The commonest cause of primary hypothyroidism is Hashimoto's thyroiditis and like all other autoimmune conditions, it is more common in females.[7] Distal paresthesias were complained by 28.6 % of patients which was similar to the study from Netherlands 29% [8] but is lower than that of study from Brazil 68.7%.[9] Muscle weakness was seen in 5.7% of patients which was similar to the study from Brazil 6.3% [9] but was lower than that of the study from Netherlands 37.5%.[8] Muscle weakness is seen mainly in the proximal muscles, symmetrically and in a non-selective manner. It is usually mild and is more pronounced in the lower limbs. The mechanism of muscle involvement in hypothyroidism remains debated. Atrophy and loss of type II fibres with intact type I fibres have been demonstrated.[10] However, it seems that the biochemical abnormalities play an important role in the pathogenesis of hypothyroid myopathy by affecting the glycolytic pathway. Predominant atrophy of type I1 fibbers which are fast in action, rich in myophosphorylase and utilize a glycolytic pathway.[10] Thus, selective atrophy of type II fibres, resulting in relative preponderance of type I fibres which are slow in action and derive energy from the oxidative cycle, might explain the weakness and sluggishness seen in hypothyroid patients.

Regarding the cognitive impairment in hypothyroidism, as assessed by Folstein MMSE , 5.7% had low score for their education level which is lower (28%) than that observed by a study on non-demented hypothyroid adults done by American Geriatrics Society.[11] The low percentage of cognitive dysfunction in the present study may be due to younger age group. None of the hypothyroid patients were clinically demented. However, the subgroup of hypothyroid patients with abnormal MMSE were very near the threshold for clinical dementia and might have reached this threshold if the hypothyroidism had persisted. Therefore it is suggested that thyroid function tests should be included in the evaluation of dementia.

Sensorineural type of deafness was seen in 5.7% of patients in the present study. An Indian study from Varanasi on audiological evaluation in hypothyroidism, found that 39% patients had an audiological proven deafness. They reported sensorineural hearing loss in 15% of cases, a conductive hearing loss in 8%, and a mixed hearing loss in 13% of cases.[12] The hearing impairment in hypothyroidism may be conductive, sensorineural, or mixed and benefits from thyroid replacement therapy.[13] The pathophysiological mechanisms of hearing loss in hypothyroidism are not totally unveiled. It may be caused in part by a general decrease in cerebral activity, a myxoedematous infiltration of the middle ear may explain loss of conduction.[14]

In the present study, the findings of BAEP included prolongation of wave V latency in hypothyroid patients. But there were no abnormalities in other waves. Similar to this, there was significant prolongation of latency of wave V in a study from Chandigarh, India [15] in which euthyroid state is achieved after treatment. There was no significant reversal in the latency after treatment. However, in another study from Delhi, India [16], there was significant improvement in the

latency of wave III after treatment. Another study from Kanchipuram, India [17] reported prolonged peak latency of wave III and V in hypothyroid patients which improved significantly after treatment. They suggested that in hypothyroid state there may be some slow conduction at the periphery. They attributed to two reasons for this, one is a delay in myelination and other is because of disturbance in the neurotransmitter mechanism.[18,19]

In the Nerve conduction studies, the sensory amplitude and conduction velocity of median nerve was significantly decreased in hypothyroid patients when compared with the control group. These findings are in consistent with that of a study from Egypt [6] and a study from Iraq [5] in which electrophysiological findings showed that median sensory latency was also prolonged. The motor conduction velocity of the median nerve in hypothyroid patients was significantly decreased when compared with the control group; these results were comparable with other studies from Egypt [6], Netherlands [8] and Iraq [5]. The motor and sensory conduction velocities from ulnar nerve also showed significant decrease in the patients group compared to control group in our study which is contrary to the study from Iraq [5], where there was no significant alteration in ulnar nerve conduction parameters. The motor conductions from common peroneal nerve showed statistically significant decreased amplitudes from both sides in our study in the patient group. The mean values of sensory conductions from sural nerve showed no significant changes which was contrary to the study from Iraq [5] where the latency was prolonged and the amplitude and conduction velocity were decreased. The conductive parameters of the tibial nerve (distal motor latency, motor conduction velocity) in hypothyroid patients were similar to that of control group, these results are same as that found in other studies from Egypt [6] and Iraq [5]. The decreased conduction velocities and amplitudes may be due to the metabolic alteration caused by hypothyroidism which affects the Schwann cell, inducing demyelination that could be segmental.[20] Primary axonal degeneration has also been shown electrophysiologically and affected initially, but structural alterations may occur later.[21]

In the present study 51.4% of patients had at least one type of electrophysiological abnormality which was similar (59%) to that observed in a study from Turkey.[22] Sensorimotor neuropathy was most commonly seen in our study (17%) whereas sensory neuropathy was reported in 44.8% by the study from Iraq.[5]

Isolated carpal tunnel syndrome (CTS) was found in 5.7% in the present study which was less than that of reported from Turkey [22] and from Netherlands.[8] Carpal tunnel syndrome (CTS) is caused by the deposition of mucinous material in the tissue surrounding the median nerve combined with hypothyroidism induced demyelination.[20]

Sural sensory mononeuropathy was seen in 11.4%, lower (25%) than that of the study from Iraq.[5] The lower incidence in our study could be due to a short duration of disease. Subclinical involvement was found in 14% of cases in this study by ENMG. Similar percentage of patients (14.3%) had subclinical involvement in the study from Brazil.[9]

In the Interpeak latency (IPL) of BAEP, there was a significant prolongation of IPL I-III, I-V in patients, but there was no prolongation in IPL of III-V. Similar to this was seen in the study from Chandigarh [15], but there was no improvement after treatment. However another study from Delhi, India [16] showed significant decrease in IPL I-III after treatment. The study from Kanchipuram [17] reported significant prolonged IPL I-V and showed improvement after treatment. The abnormalities were explained on the basis of low metabolic rate and a low body temperature of these patients.

In the present study on BAEP, there was no significant reduction in amplitudes I-Ia and V-Va in hypothyroid patients. This is contrary to that observed by the other study from Kanchipuram [17] where significantly decreased amplitudes of wave I and V were noted which improved after treatment. Another study from Delhi also showed significant improvement of amplitudes of wave I and V after treatment.[16]

However, in study from France, where BAEP was recorded and found no statistically significant differences were found between patients and controls for any of the wave latencies or IPL studied. When comparing the BAEP results obtained before and after treatment, they again found no significant changes in I, III or V latencies or in I-V or III-V IPL.[23] This could be due to variation in recording procedure, sample size and extent of dysfunction.

Limitations

This is a single centre study consisting of small number sample size. The study findings may not be generalized due to small sample size and all the patients belongs to the single geographical region. Multicentric studies with larger sample size are needed to generalize these research findings.

Conclusions

Hypothyroidism is a common condition causing neurological complications involving both central nervous system and peripheral nervous system. Hypothyroidism is one of the reversible causes of dementia. It is recommended that thyroid function tests should be included in the work up of dementia, depression, parasthesias and myopathies. BAEP evaluation suggesting that nervous system dysfunction seen in hypothyroidism is diffuse in pattern, affecting both peripheral conduction time and central conduction time.

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Legends Tables

Table 1: Comparison of Motor Nerve Conduction Between Patients and Controls

Segment of Motor Nerve	Electrophysiological Characteristic	Patients (n=35)	Controls (n=20)	p-value
Right Median Nerve	Latency (ms)	3.31 ± 0.74	3.11 ± 0.32	0.24
	Amplitude (mv)	9.05 ± 2.94	9.21 ± 2.04	0.83
	Conduction Velocity (m/s)	56.25 ± 6.95	60.77± 3.47	0.009*
Left Median Nerve	Latency (ms)	3.27 ± 0.88	2.89 ± 0.33	0.06*
	Amplitude (mv)	8.22 ± 2.4	9.25 ± 1.8	0.1
	Conduction Velocity (m/s)	57.85 ± 7.53	61.09 ± 3.76	0.08
Right Ulnar Nerve	Latency (ms)	2.36 ± 0.5	2.3 ± 0.31	0.63

	Amplitude (mv)	8.3 ± 1.8	8.16 ± 1.68	0.78
	Conduction Velocity (m/s)	59.34 ± 7.36	60.31 ± 5.3	0.6
Left Ulnar Nerve	Latency (ms)	2.4 ± 0.51	2.17 ± 0.22	0.06
	Amplitude (mv)	8.22 ± 2.62	8.33 ± 1.78	0.86
	Conduction Velocity (m/s)	58.57 ± 7.36	62.48 ± 4.78	0.03*
Right Common Peroneal Nerve	Latency (ms)	3.46 ± 0.41	3.61 ± 0.36	0.18
	Amplitude (mv)	5.61 ± 2.7	7.07 ± 1.89	0.04*
	Conduction Velocity (m/s)	46.95 ± 12.6	50.87 ± 3.17	0.18
Left Common Peroneal Nerve	Latency (ms)	3.58 ± 0.6	3.72 ± 0.38	0.35
	Amplitude (mv)	5.44 ± 2.58	6.82 ±2.16	0.04*
	Conduction Velocity (m/s)	47.06 ± 12.81	51.29 ± 3.03	0.15
Right Posterior Tibial Nerve	Latency (ms)	3.6 ± 0.62	3.41 ± 0.55	0.26
	Amplitude (mv)	9.68 ± 3.92	10.8 ±3.12	0.28
	Conduction Velocity (m/s)	47.84 ± 9.6	48.58 ± 2.29	0.73
Left Posterior Tibial Nerve	Latency (ms)	3.49 ± 0.47	3.41 ± 0.39	0.52
	Amplitude (mv)	9.51 ± 3.75	10.67±2.68	0.23
	Conduction Velocity (m/s)	48.73 ± 9.7	49.71 ± 4.33	0.61

ms: milli second; mv: milli volts. *indicates significant p-value.

Table-2: Comparison of Sensory Nerve Conduction Between Patients and Controls:

Segment of Sensory Nerve	Electrophysiological Characteristic	Patients (n=35)	Controls (n=20)	p-value
Right Median Nerve	Latency (ms)	2.6 ± 0.45	2.47 ± 0.22	0.23
	Amplitude (µv)	20.17 ±11.84	23.3±10	0.32
	Conduction Velocity (m/s)	50.83 ± 11.44	54.97 ± 4.72	0.13
Left Median Nerve	Latency (ms)	2.6 ± 0.6	2.4 ± 0.33	0.17
	Amplitude (µv)	21 ±10.9	26.97±11.14	0.05*
	Conduction Velocity (m/s)	49.83 ± 15	57.33 ±6.31	0.04*
Right Ulnar Nerve	Latency (ms)	2.02 ± 0.3	1.98± 0.16	0.58
	Amplitude (µv)	13.2 ±6.02	13.36±4.58	0.91
	Conduction Velocity (m/s)	54.1 ± 10.95	57.58 ± 3.67	0.17
Left Ulnar Nerve	Latency (ms)	2.01 ± 0.3	1.9± 0.14	0.11
	Amplitude (µv)	15.79 ±7.32	15.27±5.62	0.78
	Conduction Velocity (m/s)	54.22 ± 11.09	59.63 ±4.95	0.04*

Right Sural Nerve	Latency (ms)	2.52 ± 0.3	2.58± 0.2	0.43
	Amplitude (µv)	18.11 ±10.54	22.23±8.54	0.11
	Conduction Velocity (m/s)	50.07 ± 13.4	52.8 ± 3.6	0.37
Left Sural Nerve	Latency (ms)	2.53 ± 0.41	2.58± 0.19	0.6
	Amplitude (µv)	18.65 ±11.29	23.8±10.46	0.1
	Conduction Velocity (m/s)	51.97 ± 10.86	53.1 ± 3.98	0.65

ms: milli second; mv: milli volts. *indicates significant p-value.

Table 3: Prevalence of Neuropathy in Hypothyroid patients:

Findings	Patients (n=35)	Controls (n=20)	p-value
Sensory motor polyneuropathy	6 (17%)	0	0.13
Motor neuropathy	4 (11.43%)	0	0.30
Isolated CTS	2 (5.71%)	0	0.73
Sural sensory mononeuropathy	4 (11.43%)	0	0.30

CTS: Carpal tunnel syndrome.

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