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Research Paper

Peripheral Nerves' Involvement in Controlled and Uncontrolled Hypothyroidism: An Electrodiagnostic Study

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ABSTRACT:

BACKGROUND:

Hypothyroidism arises from reduced thyroid hormone production or activity due to dysfunction of the hypothalamic-pituitary-thyroid axis. Peripheral polyneuropathy is caused by axonal or myelin sheath damage.

OBJECTIVE:

This study aimed to explore the neurophysiological impact of hypothyroidism, not to test an intervention.

METHODS:

A cross-sectional observational study was conducted from March to December 2024, involving 50 female hypothyroid patients aged 20–75 years. Patients were divided into controlled (n = 14) and uncontrolled (n = 36) groups. All underwent motor and sensory nerve conduction studies (NCS), F-wave analysis, sural-radial amplitude ratio (SRAR), and needle EMG. Peripheral neuropathy was diagnosed based on reduced SNAP and/or CMAP amplitudes in a length-dependent pattern, while CTS was identified using standard diagnostic criteria including prolonged distal motor latency and reduced median SNAP amplitude.

RESULTS:

While age and etiology of hypothyroidism were similar between groups, disease duration was significantly shorter in uncontrolled patients ($p = 0.007$). 22 patients had peripheral neuropathy, 14 had carpal tunnel syndrome, and 14 had normal neurophysiological findings. Uncontrolled patients showed significantly reduced SNAP and CMAP amplitudes across multiple nerves ($p < 0.05$) and lower SRAR ($p < 0.001$). In the uncontrolled group, TSH was inversely associated with sural SCV ($r = –0.480$; $p = 0.004$) and positively with median DML ($r = 0.364$; $p = 0.032$).

CONCLUSION:

Despite shorter disease duration, uncontrolled hypothyroid patients showed more peripheral nerve issues possibly indicating subclinical disease. Better metabolic control improves neurophysiological outcomes. Regular thyroid hormone and nerve tests are vital for early detection and prevention. Proper hormonal management is key for metabolic health and nerve integrity.

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Iraqi Postgraduate Medical Journal, 2026; Vol.25 (2): 136-144
DOI: 10.52573/ipmj.2025.162851 *Received: July 29, 2025* *Accepted: August 4, 2025*

INTRODUCTION:

Hypothyroidism is a multifaceted endocrine disorder characterized by impaired function of the hypothalamic-pituitary-thyroid (HPT) axis, resulting in reduced synthesis, release, or biological activity of thyroid hormones (THs) at the tissue level. Its global prevalence ranges from 0.2% to 5.3% for overt hypothyroidism and reaches up to 10% for subclinical forms [1]. Notably, even with biochemical normalization of thyroid-stimulating hormone (TSH), approximately 10% to 15% of patients continue to experience residual clinical symptoms [2], suggesting possible downstream dysfunction beyond endocrine correction.

Neuromuscular manifestations are frequent and variable in hypothyroid states, with reported prevalence spanning from 10% to 80% [3,4]. Both central and peripheral components of the nervous system can be affected, resulting in a spectrum of clinical symptoms [5,6]. Cranial neuropathies, most notably sensorineural hearing loss and ophthalmopathy, are also well documented [7]. Importantly, electrophysiological studies have demonstrated that even subclinical hypothyroidism can prolong conduction times in both central and peripheral nerves [8,9]. The pathogenesis is multifactorial. The accumulation of mucopolysaccharides, such as chondroitin sulfate and hyaluronic acid, compounded by

weight gain and tissue edema, can lead to compressive neuropathy and structural nerve degeneration [4–6]. Furthermore, a reduction in thyroid hormone availability may precipitate primary axonal degeneration, characterized by axonal shrinkage, neurofilament disintegration, and breakdown of the axolemma [10,11]. Electrophysiologically, common findings include reduced or absent sensory nerve action potentials, mildly decreased conduction velocities, and prolonged distal latencies. A subset of patients presents with neuropathic pain despite normal nerve conduction studies (NCS), suggesting the involvement of small fiber neuropathy. This observation is supported by advanced diagnostic tools, including intraepidermal nerve fiber density measurement, laser Doppler flare imaging, and corneal confocal microscopy [12].

This study aims to assess how hypothyroidism affects peripheral nerve function through electrodiagnostic methods such as NCS and electromyography (EMG). Additionally, it compares the electrophysiological results between hypothyroid patients with controlled and uncontrolled metabolic states, providing possible insights into how disease severity and hormonal regulation influence nerve function.

METHOD:

Between March 2024 and December 2024, this cross-sectional observational study was conducted at the Neurophysiology Unit of Baghdad Teaching Hospital/Medical City and the Neurophysiology Department at Al-Imamian Al-Kadhimyian Medical City in Baghdad. This study was conducted following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines to ensure transparent reporting [13].

Participants and Study Design

The study included fifty female patients diagnosed with hypothyroidism, confirmed by a senior endocrinologist. Participants' ages ranged from 20 to 75 years. The duration of hypothyroidism varied; some were newly diagnosed, while others had the condition for up to 25 years. Patients with known causes of peripheral neuropathy (e.g., diabetes, B12 deficiency), renal failure, pregnancy, malignancy, neuromuscular disorders, alcoholism, or use of neuropathy-inducing drugs (e.g., chemotherapy, isoniazid, phenytoin, metronidazole) were excluded. Patients were grouped based on thyroid status (TSH and fT4 levels): 36 with uncontrolled hypothyroidism and 14 with controlled hypothyroidism.

A total of 50 female patients were enrolled using a purposive sampling method. The decision to include only female participants was based on the higher prevalence of hypothyroidism among women, as well as to eliminate gender-related variability in neurophysiological parameters. No formal sample size calculation was performed; however, the sample size was guided by prior similar cross-sectional studies and logistical feasibility.

Clinical and Diagnostic Assessment

Each participant completed a comprehensive medical history and a detailed neurological exam to check for signs and symptoms of hypothyroidism and peripheral neuropathy. Thyroid function was evaluated using serum biochemical tests to measure free T4 (fT4) and TSH levels, which are crucial markers for detecting thyroid disorders and assessing treatment success. These hormones are interconnected through a log-linear feedback loop, and population-specific reference ranges are used to accurately interpret TSH values as a primary indicator of thyroid health. The established reference ranges for these tests were as follows: TSH ranged from 0.4 to 4.0 milli-international units per liter (mIU/L), free T4 ranged from 0.7 to 1.9 nanograms per deciliter (ng/dL), and free triiodothyronine (free T3) ranged from 3.0 to 7.0 picograms per milliliter (pg/mL).

Electrophysiological studies were performed using the Key Point EMG system (Medtronic, Denmark) and a Nihon Kohden electromyography unit (Japan) to assess neuromuscular function. Three types of electrodes were used during testing: a grounding electrode (H658NM-550B, disk type, DK-270, Denmark), a bipolar surface stimulating electrode (H690NM-317Y3, DK-2740, Denmark), and a surface recording electrode (H690NM-317Y3, DK-2740, Denmark). During testing, patient skin temperature was maintained between 32°C and 34°C to ensure consistent nerve conduction measurements, while ambient room temperature was kept between 25°C and 28°C.

Nerve Conduction Studies and Electromyography

Nerve conduction and EMG studies were conducted per Preston and Shapiro's protocol [14]. Sensory testing included bilateral median, ulnar, right radial, and sural nerves. Motor studies assessed median, ulnar, peroneal, and tibial nerves, with recordings from corresponding hand and foot muscles. F-waves were recorded from the right ulnar and tibial nerves. The sural/radial

amplitude ratio (SRAR) was also calculated to compare distal sensory responses. Concentric needle EMG was performed using a Micromed device (DIN 42802, Italy), with recordings from upper and lower limb muscles, including deltoid, triceps, brachioradialis, and tibialis anterior. Assessed parameters included sensory and motor latencies, conduction velocities, sensory nerve action potential amplitude (SNAP) amplitude, and minimum F-wave latency. Standardized electrophysiological settings were used: motor studies employed a 10 Hz–10 kHz filter, 5 ms/div sweep, and 5 mV/div sensitivity; sensory studies used a 20 Hz–2000 Hz filter, 2 ms/div sweep, and 20 µV/div sensitivity. motor unit action potentials (MUAPs) were analyzed at rest and during contractions, with a gain of 200 µV/div and a 20 ms/div sweep. F-waves were tested at 200 µV/div, 5–10 ms/div sweep, and a 16 Hz–16 kHz filter, averaged over ten trials using automatic waveform analysis.

Nerve conduction studies and EMG were performed using standard clinical EMG systems (Medtronic and Nihon Kohden). Skin temperature was maintained between 32°C–34°C, and room temperature was controlled at 25°C–28°C. Standard filter and gain settings were applied across sensory and motor studies. All conduction velocity values were temperature-corrected and within our laboratory's validated normative range. Motor conduction velocities exceeding 64 m/s were observed in some upper limb nerves and are considered physiologically plausible, particularly in younger patients or shorter limb segments.

Reference values for sensory and motor nerve conduction studies and F-wave latencies were based on lab-specific normative data derived from a healthy local population and consistent with established international standards [14]. Abnormalities were defined as values falling below the 5th percentile or above the 95th percentile of these norms. SNAP amplitudes < 10 µV (for sural and radial nerves) or CMAP amplitudes < 5 mV (for median and tibial nerves) were considered pathological. SRAR < 0.21 was used as a diagnostic cutoff for early sensory neuropathy. These thresholds were not age- or sex-adjusted but were applied uniformly, as all participants were female and within a relatively similar adult age range.

Statistical Analysis

The Statistical Package for Social Sciences (SPSS), version 25 (IBM Corporation, USA), was used for all statistical analyses. An independent Student's t-test was used to assess the quantitative data, which were displayed as mean ± standard deviation (SD). The Mann-Whitney U test was used to provide nonparametric, non-normally distributed data as median and range. The chi-square test was used to assess categorical variables, which were represented as median and range. Two-tailed Pearson's correlation analysis was used to evaluate the correlations between various quantitative variables. All tests were deemed statistically significant if the p-value was below 0.05.

Ethical Statement

The study was approved by the Iraqi Board for Medical Specialization (Order #240: 22/1/2023). Prior to participation, all subjects received a comprehensive explanation of the study's objectives, methodology, and procedures. Written informed consent was obtained from each participant, ensuring voluntary participation in accordance with ethical guidelines and the principles outlined in the Declaration of Helsinki.

RESULTS:

***Demographic* data**

The demographic information of the study population is presented in **Table 1**. Age and the cause of hypothyroidism did not significantly differ between the two groupings. Nonetheless, there was a significant difference ($p = 0.007$) in the length of the illness between the two groups. We used EMG only to exclude cases of myopathy and the results of the examination were not included in the comparison between the two groups.

Table 1: Demographic data of the study population.

Characteristic	Metabolic control		p-value
	Yes (n=14)	No (n=36)	
Age, years			
Mean±SD	41.64±12.67	43.33±15.07	0.712
Disease duration, months			
Mean±SD	96.14±99.58	43.39±67.65	
Median	66.0	12.0	0.007
Range	2-300	1-276	
Hypothyroid cause			
Idiopathic	14(100%)	33(91.67%)	0.368
Thyroidectomy	0(0%)	3(8.33%)	

Thyroid Function Tests

As demonstrated in Table 2, the metabolically uncontrolled individuals had considerably higher TSH levels than the controlled group ($p < 0.001$), while the uncontrolled group had significantly lower levels of fT4 and T3 ($p = 0.028$ and $p = 0.023$, respectively).

Table 2: Thyroid function tests of the study population.

Characteristic	Metabolic control		p-value
	Yes (n=14)	No (n=36)	
TSH, mIU/L			
Mean±SD	4.50±3.46	10.60±9.90	
Median	3.60	7.91	<0.001
Range	1.31-15.37	0.01-45.67	
fT4, ng/dL			
Mean±SD	13.65±2.28	11.91±2.49	0.028
T3, ng/dL			
Mean±SD	1.83±0.32	1.47±0.51	0.023

Neurophysiological Data

Out of the total 50 hypothyroid patients, 14 patients showed normal neurophysiologic data, 22 showed evidences for peripheral neuropathy (3 of them were biochemically controlled and 19 were metabolically uncontrolled), one of the cases was of demyelinating type and the rest of cases were mild axonal sensorimotor polyneuropathy affecting mainly the sensory fibers in a length dependent pattern. The rest 14 hypothyroid patients had CTS, 3 of them were biochemically controlled and 11 were metabolically uncontrolled as shown in Table 3.

Table 3: Electrodiagnostic results of the study population.

Final EDx diagnosis	Hypothyroid patients	
	Metabolically-controlled (n=14)	Non-biochemically controlled(n=36)
Normal	8	6
PNP	3	19
CTS	3	11

The amplitudes of SNAP of both the ulnar ($p < 0.001$), left median ($p = 0.006$), radial ($p < 0.001$), and sural ($p = 0.008$) nerves were considerably lower in patients with metabolically uncontrolled disorders than in the control group. Similarly, the SRAR of metabolically uncontrolled patients was considerably lower than that of the controlled group ($p < 0.001$). According to Table 4, there were no appreciable variations between the two subgroups for the remaining sensory conduction research measures.

Table 4: Sensory Nerve conduction study.

Nerve/Parameter		Metabolic control		p-value
		Yes (n=14)	No (n=36)	
RMed	**SPL, ms**	3.3±0.46	3.26±0.52	0.798
	SNAP amplitude, µV	36.25±15.07	28.71±11.43	0.069
	SCV, m/s	56.0±12.81	54.01±9.86	0.770
LMed	**SPL, ms**	3.28±0.74	3.22±1.18	0856
	SNAP amplitude, µV	38.13±16.40	25.77±12.42	0.006
	SCV, m/s	58.0±12.81	57.01±9.86	0.771
RUln	**SPL, ms**	2.72±0.35	2.85±0.45	0.305
	SNAP amplitude, µV	41.04±9.33	25.02±9.74	<0.001
	SCV, m/s	64.93±7.98	9.34±11.95	0.627
LUln	**SPL, ms**	2.64±0.31	2.60±0.42	0.758
	SNAP amplitude, µV	35.96±10.12	22.83±8.69	<0.001
	SCV, m/s	60.93±7.95	9.24±11.94	0.629
Radial	**SPL, ms**	2.57±0.54	2.30±1.15	0.417
	SNAP amplitude, µV	27.63±9.99	12.53±6.59	<0.001
	SCV, m/s	60.71±5.74	60.64±12.21	0.982
Sural	**SPL, ms**	2.80±0.64	3.15±0.68	0.113
	SNAP amplitude, µV	19.45±10.94	11.63±7.93	0.008
	SCV, m/s	58.64±5.42	52.84±12.60	0.105
Sural/Radial amplitude ratio				
Mean±SD		1.14±0.92	0.31±0.21	
Median		1.01	0.2	<0.001
Range		0.2-3.3	0.11-1.0	

In comparison to the managed group, the metabolically uncontrolled patients had substantially lower median and ulnar CMAP amplitudes on both sides ($p = 0.026$, $p < 0.001$, $p = 0.007$, and $p = 0.010$, respectively). However, as shown in Table 5, there were no appreciable variations between the two groups in the distal motor latencies (DMLs) or motor conduction velocities (MCVs) of the left and right median and ulnar nerves.

Table 5: Upper Limb Motor Nerve conduction study.

Nerve/Parameter		Metabolic control		p-value
		Yes (n=14)	No (n=36)	
RMed	**DML, ms**	3.5±1.09	3.26±0.54	0.301
	CMAP amplitude, mV	8.54±2.19	8.85±3.68	0.026
	MCV, m/s	64.95±9.92	60.18±11.32	0.175
LMed	**DML, ms**	3.07±0.65	3.29±0.92	0.419
	CMAP amplitude, Mv	9.29±1.8	6.66±2.36	<0.001
	MCV, m/s	66.95±9.92	65.18±11.32	0.179
RtUln	**DML, ms**	2.25±0.37	2.22±0.49	0.839
	CMAP amplitude, mV	8.68±1.93	6.87±2.07	0.007
	MCV, m/s	63.59±10.15	59.65±9.70	0.208
	F-wave, ms	24.79±1.86	23.31±2.85	0.080
LUln	**DML, ms**	2.27±0.35	2.32±0.64	0.798
	CMAP amplitude, mV	8.39±1.40	6.98±1.74	0.010
	MCV, m/s	25.79±1.86	26.31±2.85	0.085

In relation to the lower limb nerves, Table 6 illustrates that the metabolically uncontrolled patients' CMAP amplitudes of left tibial as well as right peroneal were considerably lower in relation to the lower limb nerves of those of the controlled group. The metabolically uncontrolled patients' left tibial and right peroneal CMAP amplitudes were considerably lower than those of the controlled group ($p = 0.008$, $p = 0.004$, respectively). Distal motor latencies (DML) and motor conduction velocities (MCV) did not, however, differ dramatically between the groups.

Table 6: Lower Limb Motor Nerve conduction study.

Nerve/Parameter		Metabolic control		p-value
		Yes (n=14)	No (n=36)	
RTib	DML, ms	3.54±1.19	3.83±1.03	0.409
	CMAP amplitude, mV	5.87±1.92	5.41±2.69	0.569
	MCV, m/s	51.21±7.67	50.35±8.42	0.742
	F-wave, ms	46.85±4.50	44.27±9.58	0.342
LTib	DML, ms	3.49±1.27	3.99±1.64	0.319
	CMAP amplitude, mV	7.23±2.39	4.95±2.65	0.008
RtPer	DML, ms	3.31±0.91	3.55±1.12	0.477
	CMAP amplitude, mV	4.01±1.47	2.89±1.03	0.004
	MCV, m/s	51.24±7.56	51.35±8.05	0.965
LPer	DML, ms	3.26±0.89	3.57±0.99	0.316
	CMAP amplitude, mV	2.85±0.91	2.85±1.09	0.997

Correlation Analysis

In biochemically controlled patients, disease duration positively correlated with median DML (r = 0.549, p = 0.024), and serum T3 levels correlated with MCV (r = 0.671, p = 0.012). In uncontrolled patients, TSH levels were positively associated with DML (r = 0.364, p = 0.032; Figure 1) and inversely with sural SCV (r = –0.480, p = 0.004; Figure 2).

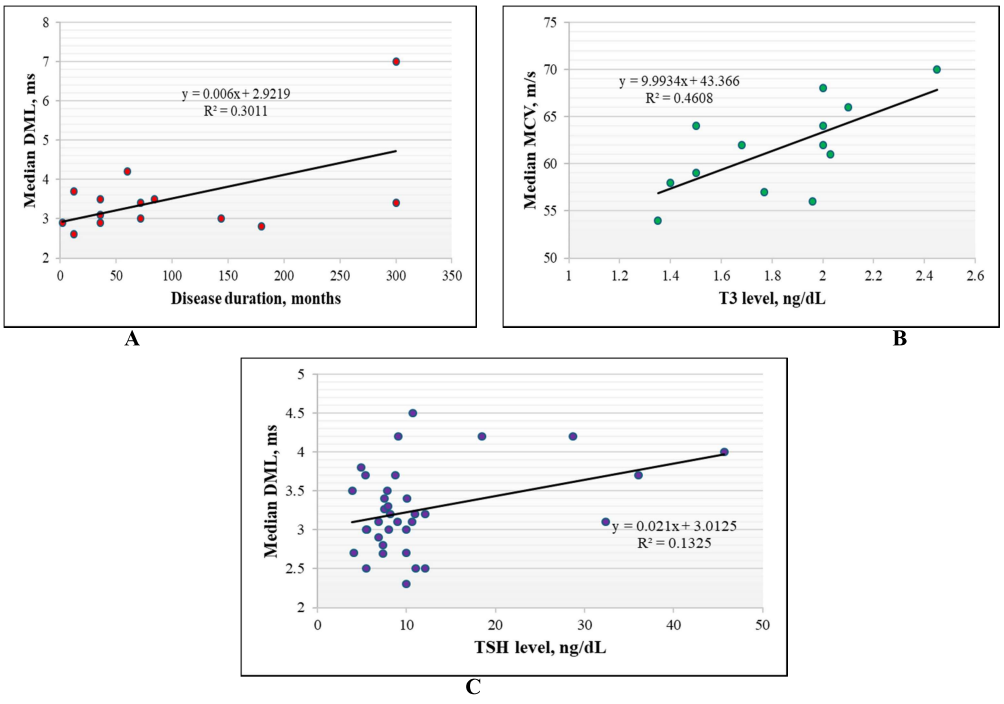

Figure 1: Scatter plots with regression lines.
A: Relationship between disease duration and median distal motor latency (DML) in biochemically controlled hypothyroid patients.
B: Association between serum T3 levels and median motor conduction velocity (MCV) in biochemically controlled hypothyroid patients.
C: Correlation between TSH levels and median DML in metabolically uncontrolled hypothyroid patients, illustrated using a scatter diagram and regression alignment.

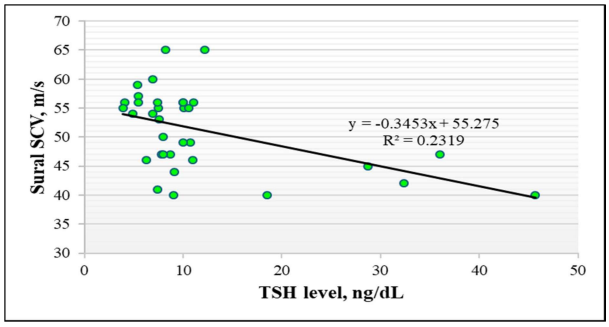

Figure 2: The association between TSH level and sural sensory conduction velocity (SCV) in individuals with metabolically uncontrolled hypothyroidism is displayed using a scatter graph and a regression in line.

DISCUSSION:

In this study, no significant differences were found between the biochemically controlled and uncontrolled hypothyroid patient groups in terms of age or the underlying etiology of hypothyroidism. However, disease duration differed significantly, likely reflecting a shorter period of illness and earlier diagnosis in the controlled group, allowing for more timely metabolic management. As expected, patients with uncontrolled hypothyroidism demonstrated higher serum TSH levels and lower concentrations of T3 and free T4, consistent with the classical biochemical profile of primary hypothyroidism, which is well-established as a diagnostic hallmark [15].

Electrophysiological abnormalities suggestive of peripheral neuropathy were identified in a substantial proportion of patients. Specifically, 22 individuals met the criteria for sensorimotor neuropathy based on a sural/radial amplitude ratio (SRAR) of less than 0.21 [14], while another 14 patients fulfilled the clinical and electrodiagnostic criteria for CTS [16]. Nerve conduction studies revealed that patients with metabolically uncontrolled hypothyroidism exhibited significantly reduced CMAP and SNAP amplitudes in both upper and lower limbs, as well as lower SRAR values, compared to their biochemically controlled counterparts. A cross-sectional prospective study by Gupta et al. in India, involving 120 participants aged 20–50 years, reported reduced sensory nerve amplitudes (median and sural), prolonged latencies, and decreased sensory conduction velocities. Motor conduction velocities and amplitudes of the median and tibial nerves were also reduced in hypothyroid patients compared to healthy controls [17]. Similarly, a study from Egypt on 30 hypothyroid patients observed significantly decreased SNAP amplitudes in the median, ulnar, and sural nerves, as well as reduced CMAP amplitudes in the median, tibial, ulnar, and common peroneal nerves [18]. Another Indian study on newly diagnosed female patients found reduced ulnar SNAP amplitudes and SCVs, prolonged sensory latencies, and impaired conduction across median, tibial, and sural nerves, with preservation of ulnar motor function [19]. Additional confirmation comes from a cross-sectional study in North Macedonia involving 78 subjects, which showed that median nerve SNAP and CMAP amplitudes were significantly reduced in patients with primary hypothyroidism compared to euthyroid controls [20]. Interestingly, some patients in this study (28%) displayed no electrophysiological abnormalities, despite clinical hypothyroidism. This aligns with findings by Jalilzadeh et al., who reported a higher prevalence than the 13.4% reported by Abdelazeem et al. [18].

A subset of patients in our study reported neuropathic symptoms despite normal findings on standard NCS and EMG. This may be explained by small fiber neuropathy, which primarily affects thinly myelinated Aδ and unmyelinated C fibers that are not assessed by conventional electrodiagnostic techniques. Advanced diagnostic methods, such as intraepidermal nerve fiber density measurement from skin biopsy, corneal confocal microscopy, or laser Doppler flare imaging, could provide additional insights into early or subclinical neuropathy in hypothyroid patients. Future studies incorporating these modalities are recommended.

Our study observed sensorimotor neuropathy in 44% of hypothyroid patients. The incidence of CTS in our cohort was 28%, comparable to previous studies where CTS coexisted in 23.7% to 30% of hypothyroid patients [21,22]. Notably, this study demonstrated a statistically significant positive correlation between disease duration and

median DML in biochemically controlled patients. In metabolically uncontrolled patients, a positive correlation between TSH levels and median DML was observed. Interestingly, in our study, TSH levels were inversely correlated with sural SCV. These variable findings highlight the need for further longitudinal investigations to better understand the interplay between thyroid hormone levels and peripheral nerve function.

The observed reductions in SNAP and CMAP amplitudes among uncontrolled patients, particularly in the median, ulnar, and sural nerves, are not only statistically significant but also clinically relevant. These changes suggest early axonal involvement, which may precede overt neuropathic disability and could be reversible with timely thyroid hormone correction.

This study underlines the importance of maintaining optimal thyroid hormone levels in hypothyroid patients to protect nerve function. Electrodiagnostic abnormalities were more evident in those with poor metabolic control. Regular neurophysiological assessments, especially in patients with high TSH or ongoing symptoms, can detect peripheral neuropathy early, possibly reversible.

Limitations

The small sample size, especially in the biochemically controlled group, may limit statistical power and generalizability. The cross-sectional design prevents causal inferences or understanding neuropathy progression; longitudinal studies are needed. Despite efforts to exclude factors like diabetes and vitamin B12 deficiency, subclinical conditions or unmeasured lifestyle factors could influence outcomes. Although strict exclusion criteria were applied to minimize known causes of neuropathy, the influence of unmeasured or subclinical confounders cannot be ruled out. Borderline vitamin B12 deficiency, undiagnosed autoimmune diseases, or sedentary lifestyle may have subtly impacted nerve function in some participants. Additionally, technical factors such as minor inconsistencies in electrode placement, limb length, or patient compliance during EMG testing could affect amplitude values, though all procedures followed standardized protocols under expert supervision.

CONCLUSION:

This study highlights the significant impact of metabolic control on peripheral nerve health in patients with hypothyroidism. Those with poorly managed hypothyroidism showed more severe neurophysiological impairments, while patients with well-managed thyroid function had better nerve conduction results. Future studies should adopt a longitudinal design incorporating serial electrodiagnostic assessments to evaluate the trajectory of nerve function following thyroid hormone optimization. Such designs could determine whether improved metabolic control leads to functional nerve recovery or merely halts further deterioration. Including a healthy control group would also provide a clearer baseline for comparison and strengthen interpretation of neurophysiological changes attributable to hypothyroidism.

Declarations

Ethics approval and consent to participate: The study was approved by the Iraqi Board for Medical Specialization (Order #240: 22/1/2023). All participants provided written informed consent prior to enrollment, in accordance with the principles outlined in the Declaration of Helsinki.

Consent for publication: Written informed consent was obtained from all participants for the anonymized data to be used in research and publication.

Availability of data and material: The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests: The authors declare that they have no competing interests.

Funding: No specific funding was received for this study.

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