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SUBCLINICAL PERIPHERAL NERVE AFFECTION IN CASES OF SUBCLINICAL HYPOTHYROIDISM

By

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ABSTRACT

Background: One of the most frequent etiologies of endocrinal polyneuropathy is the hypothyroidism, that can cause axonal and demyelinating polyneuropathies.

Objective: To evaluate Subclinical affection of peripheral nerve among patient with subclinical hypothyroidism.

Patients and methods: Fifty consecutives recently diagnosed sub clinical hypothyroid patients referred from outpatients' clinic of Internal Medicine Department to Neurophysiology unit of Neurology Department of Al-Azhar University hospital in new Damietta, and fifty age and gender matched euthyroid healthy persons The electrophysiological study of all studied groups was done including motor and sensory nerve conduction studies of both median, ulnar, peroneal and sural nerves.

Results: As regard to motor conduction study. There was a significant deference between both groups as regard of CMAP amplitude of both peroneal nerves, conduction velocity of both median and both peroneal nerves. Regarding to sensory conduction study there was significant deference between both groups as regard of of sensory terminal latency, SNAP amplitude of both median and sural nerves. There was a significant negative correlation between TSH serum level and each of conduction velocity of both median, both peroneal, left ulnar nerves, compound motor unit action potential amplitude of both peroneal nerves, and sensory nerve action potential amplitude of right sural nerve. There was a significant +ve correlation between the TSH serum level and terminal latency of both peroneal and right sural nerves.

Conclusion: Subclinical hypothyroidism can be considered as risk factor of peripheral nerve affection as in cases of hypothyroidism

Keywords: subclinical hypothyroidism, electrophysiological study, endocrinal polyneuropathy.

INTRODUCTION

One of the most frequent etiologies of endocrinal polyneuropathy is the hypothyroidism that can cause axonal and demyelinating polyneuropathies. The diagnosis of neuropathy can be confirmed by electrophysiological examination in most of them, and the clinical manifestation occasionally observed in hypothyroid patients. Subclinical changes in conduction of motor and sensory fibers are relative common., The influence of subclinical hypothyroidism on peripheral nerves is undefined *(Abdelazeem et al., 2017)*. The electrophysiological test is very sensitive test for diagnosis of neuropathy among hypothyroid patients *(Waghmar et al., 2016)*.

The pain of polyneuropathy affects the life quality of patients, and the pain killer drugs remain disappointing *(Jaiswal and Dhankad, 2020)*. Treatment of the possible cause was considered as the appropriate therapy. Hormonal treatment can reverse the clinical and electrophysiological finding of neuropathy

in cases with subclinical hypothyroidism *(Jaiswal and Dhankad, 2020)*. The overall prevalence of subclinical hypothyroidism is 4-10% *(ALEidan et al., 2018)*. The subclinical hypothrodism is common in elderly and affecting females more than males.The prevelance among genral population is 2.5-10%, and the prevalence of peripheral neuropathy among the hypothyroidism is 10-70% *(Silva and Costa, 2013)*. The possible mechanism of neuropathy is focal demyelination or axonal degeneration. The subclinical hypothyroidism can be a possible. etiology of neuropathy and hormonal replacement therapy can improve this condition *(Jaiswal and Dhankad, 2020)*.

The most prevalent disorder preceding the manifest hypothyroidism is subclinical hypothyroidism. This can be diagnosed when normal titraiodothyroxin (T3) & free thyroxin (T4) serum level but TSH of high serum level. The thyroid hormons have a role in myelination, development and growth of neurons *(Jaiswal and Dhankad, 2020)*.

The electrophysiological studies showed affected measurements in patients with subclinical hypothyroidism, and the studies in assessment of nervous system (central and peripheral) in patients with sub clinical hypothyroidism is few and contra- versed *(Jaiswal and Dhankad, 2020)*.

The subclinical hypothyroidism can be defined as asymptomatic or oligo-symptomatic condition with high thyroid stimulating serum level and normal free thyroxin serum level. Sub clinical hypothyroidism representation is common in outpatient clinic by a specific symptoms like paresthesia, sexual dysfunction, chronic fatigue, psychiatric symptoms, myalgia, and mild cognitive impairment or asymptomatic *(Silva and Costa, 2013)*.

The aim of the study was to evaluate the subclinical peripheral nerve affection among patients with subclinical hypothyroidism.

PATIENTS AND METHODS

Fifty consecutives recently diagnosed sub clinical hypothyroid patients referred from outpatients' clinic of Internal Medicine Department to Neurophysiology unit of Neurology Department of Al-Azhar University Hospital in new Damietta, and 50 age and gender matched euthyroid healthy persons attended to the out patients as relatives to our stuff and students. the study was ethically approved by the Institutional Review Board of Damietta Faculty of Medicine, Al-Azhar University, and we excluded from our study the subject who have systemic diseases causing neuropathy, like: diabetes, renal impairment, manifest thyroid dysfunction, liver impairment, dyslipidemia, rheumatoid arthritis, malignancy, history of alcoholism, vitamin deficiency, and drug induced neuropathy like chemotherapy. The demographic criteria were studied including the age and gender.

The test of thyroid function was included measurement of serum TSH, free T3, and free T4, Subclinical hypothyroidism (SCH) was defined when serum thyroid-stimulating hormone (TSH) level above the upper limit of normal despite normal levels of serum free thyroxine) *(Silva and Costa, 2013)*. The subclinical hypothyroidism was diagnosed when TSH level exceeded the normal

range) 0.27-4.5 uIU/ mL), and FT4 within normal range (0.93-1.7ng/dL).

The electrophysiological study of subjects was conducted using Nihon Kohden Machine, model UT-0800 J, Box BOARD (2CH) for JB-942BK (Japan).

Terminal latency (TL), conduction velocity (CV) and amplitude of Compound Motor Action Potential (CMAP) through electrical stimulation of ulnar and median nerves using supramaximal nerve stimulation delivered by bipolar electrical stimulator (at wrist and elbow), and recording the evoked response form abductor digiti minimi and abductor policies brevis using two service electrode (active and reference) the active on the belly of the muscle and the reference 3 cm distal the active electrode on metacarpophalangeal joint and the ground electrode placed between the stimulator and recording electrodes.

Terminal latency, conduction velocity and (CMAP) amplitude through electrical stimulation of peroneal nerve using supramaximal nerve stimulation delivered by bipolar electrical stimulator (at ankle and neck of fibula) and recording the evoked response form extensor digitorium brevis muscle using two service electrode (active and reference) the active placed 7 cm anterior to lateral malleolus and the reference 3 cm distal the active electrode on metacarpophalangeal joint and the ground electrode placed between the stimulator and recording electrodes.

Sensory terminal latency (STL), and (SNAP) through electrical stimulation of ulnar and median nerves using supramaximal nerve stimulation delivered by bipolar electrical stimulator at wrist and recording the evoked response form the little and middle fingers using two ring electrodes (active and reference) the active on 1st inter phalangeal joint and the reference on 2nd inter phalangeal joint and the ground electrode placed between the stimulator and recording electrodes. and electrical stimulation of sural nerve using supramaximal nerve stimulation delivered by bipolar electrical stimulator at the back of leg 12 cm above the lateral malleuolus and recording the evoked response form are behind the lateral malleuolus using two surface electrodes 3cm between the active and reference, and the ground electrode placed between the stimulator and recording electrodes.

The neuropathy was diagnosed when the measurement exceeded the normal values according to Preston and Shapiro 2005 *(Abdelazeem et al., 2017)*:

1. Motor median distal latency, conduction velocity and amplitude are ≤4.4ms, ≥ 49m/s & ≥4mv respectively.
2. Motor ulnar distal latency, conduction velocity and amplitude were

≤3.3ms, ≥ 49m/s & ≥6mv respectively.

3. Motor peroneal distal latency, conduction velocity and amplitude were ≤6.5ms, ≥ 42m/s & ≥2mv respectively.
4. Sensory median distal peak latency, conduction velocity and amplitude were ≤3.5ms, ≥ 50 m/s & ≥20uv respectively.
5. Sensory ulnar distal peak latency, conduction velocity and amplitude were ≤3.1ms, ≥ 50 m/s & ≥17uv respectively.

6. Sensory sural distal peak latency, conduction velocity and amplitude were ≤4.4ms, ≥ 40 m/s & ≥ 6uv respectively.

Statistical analysis: The collected data were analyzed using the SPSS vesion 25.

The data were expressed as means and standerd deviation. Numerical data were compared by two sample Z test for means. Pearson Correlation Coefficient test were used for study the correlation between TSH level and parameters of nerve conduction study within the subclinical hypothyroidism group. And to identify the predictor of subclinical neuropathy we were used regression analysis test and the risk of estimation for detection of the risk factor of sub clinical neuropathy. P> 0.05 was considered significant.

RESULTS

Both groups showed no significant differences as regard age distribution and T4 serum level, but TSH serum level showed significant differences between both groups.

As regard to motor conduction study, there was a significant difference between both groups as regard of CMAP amplitude of both peroneal nerves, conduction velocity of both median and both peroneal nerves **(Table 1)**.

Table (1): Comparison between SCH and euthyroid groups as regard to motor conduction study of both median, ulnar and peroneal nerves

Groups / parameters	Groups (N50)	Mean	Std. Deviation	Z	Sig.
RT_MEDIAN_DML (ms)	SCH	4.1563	0.77502	1.3592	0.1746
	EU	3.6367	0.38728		
LT_MEDIAN_DML (ms)	SCH	3.8397	0.69409	0.4512	0.6512
	EU	3.6467	0.42567		
RT_MEDIAN_CV (m/s)	SCH	49.9833	6.04079	-3.5411	0.0004
	EU	60.5000	5.27682		
LT_MEDIAN_CV (m/s)	SCH	47.2667	8.67391	-3.3586	0.0008
	EU	60.5000	5.27682		
RT_MEDIAN_CMAP (mv)	SCH	5.1530	1.26763	-0.7913	0.4292
	EU	5.4500	0.88034		
LT_MEDIAN_CMAP (mv)	SCH	4.9597	1.52172	-1.2187	0.2239
	EU	5.6000	0.84649		
RT_ULNAR_DML (ms)	SCH	2.8390	0.42783	0.0000	1.0000
	EU	2.8390	0.42783		
LT_ULNAR_DML (ms)	SCH	2.8627	0.45098	0.0000	1.0000
	EU	2.8627	0.45098		
RT_ULNAR_CV (m/s)	SCH	52.9000	5.42249	0.0000	1.0000
	EU	52.9000	5.42249		
LT_ULNAR_CV (m/s)	SCH	54.4000	6.65194	0.0000	1.0000
	EU	54.4000	6.65194		
RT_ULNAR_CMAP (mv)	SCH	6.2200	1.20413	0.0000	1.0000
	EU	6.2200	1.20413		
LT_ULNAR_CMAP (mv)	SCH	6.3167	1.16444	0.0000	1.0000
	EU	6.3167	1.16444		
RT_PERONEAL_DML (ms)	SCH	6.3700	1.18682	1.5673	0.1167
	EU	5.3933	0.62197		
LT_PERONEAL_DML (ms)	SCH	6.3210	0.94785	1.6277	0.1036
	EU	5.3600	0.63333		
RT_PERONEAL_CMAP (mv)	SCH	2.0293	0.92750	-1.7501	0.0402
	EU	3.1567	0.64470		
LT_PERONEAL_CMAP (mv)	SCH	2.0470	1.06220	-2.1809	0.0145
	EU	3.3967	0.73695		
RT_PERONEAL_CV (m/s)	SCH	42.9333	9.47932	-2.1324	0.0165
	EU	53.5667	4.73201		
LT_PERONEAL_CV (m/s)	SCH	40.9667	11.91488	-3.1297	0.0018
	EU	53.9000	4.42056		

SCH =subclinical hypothyroidism. EU= euthyroid

As regard to sensory conduction study, there was a significant difference between

both groups as regard of sensory terminal latency, SNAP amplitude of both median and sural nerves **(Table 2)**.

Table (2): Comparison between SCH and euthyroid groups as regard to sensory conduction study of both median, ulnar and sural nerves

Groups / Parameters	Groups (N:50)	Mean	Std.Deviation	Z	Sig.
RT_MEDIAN_STL (ms)	SCH	3.5073	0.64461	4.638	<0.0001
	EU	2.9043	0.38810		
LT_MEIAN_STL (ms)	SCH	3.4623	0.58804	4.519	<0.0001
	EU	2.8877	0.36074		
RT_MEDIAN_SNAP (uv)	SCH	19.7333	5.33591	-7.855	<0.0001
	EU	24.7267	3.21933		
LT_MEDIAN_SNAP (uv)	SCH	21.4333	5.05612	-5.319	<0.0001
	EU	24.7600	3.22743		
RT_ULNAR_STL (ms)	SCH	2.8493	0.55092	0.000	1.0000
	EU	2.8493	0.55092		
LT_ULNAR_STL (ms)	SCH	2.8243	0.46972	0.000	1.0000
	EU	2.8243	0.46972		
RT_ULNAR_SNAP (uv)	SCH	18.4167	2.74202	0.000	1.0000
	EU	18.4167	2.74202		
LT_ULNAR_SNAP (uv)	SCH	18.2433	2.82790	0.000	1.0000
	EU	18.2433	2.82790		
RT_SURAL_STL (ms)	SCH	4.4067	1.24372	5.176	<0.0001
	EU	3.3533	0.61908		
LT_SURAL_STL (ms)	SCH	4.5533	1.47127	4.636	<0.0001
	EU	3.3900	0.61102		
RT_SURAL_SNAP (uv)	SCH	5.3167	2.65617	-4.358	<0.0001
	EU	6.5833	1.35852		
LT_SURAL_SNAP (uv)	SCH	5.4700	2.57657	-3.327	0.0009
	EU	6.5133	1.43881		

There is a significant -ve correlation between the TSH serum level and each of the following: 1) Conduction velocity of both median, both peroneal, and left ulnar nerves. 2) Compound motor unit action potential amplitude of both peroneal nerves. 3) Sensory nerve action potential amplitude of right sural nerve. There was a significant +ve correlation between the TSH serum level and terminal latency of both peroneal and right sural nerves **(Table 3)**.

Table (3): Correlation between TSH level and parameters of nerve conduction study within the subclinical hypothyroidism group

TSH / Parameters	**Pearson Correlation**	**Sig. (2-tailed)**
RT_MEDIAN_DML	0.177	0.35
LT_MEDIAN_DML	0.008	0.967
RT_MEDIAN_CV	-.586-**	0.001
LT_MEDIAN_CV	-.390-*	0.033
RT_MEDIAN_CMAP	0.005	0.981
LT_MEDIAN_CMAP	-.208-	0.271
RT_ULNAR_DML	-.329-	0.076
LT_ULNAR_DML	-.263-	0.16
RT_ULNAR_CV	-.179-	0.343
LT_ULNAR_CV	-.395-*	0.031
RT_ULNAR_CMAP	-.265-	0.156
LT_ULNAR_CMAP	-.360-	0.051
RT_PERONEAL_DML	.569**	0.001
LT_PERONEAL_DML	.484**	0.007
RT_PERONEAL_CMAP	-.613-**	0
LT_PERONEAL_CMAP	-.365-*	0.047
RT_PERONEAL_CV	-.471-**	0.009
LT_PERONEAL_CV	-.512-**	0.004
RT_ULNAR_STL	0.212	0.261
LT_ULNAR_STL	-.163-	0.389
RT_ULNAR_SNAP	-.228-	0.225
LT_ULNAR_SNAP	-.073-	0.701
RT_MEDIAN_STL	0.147	0.439
LT_MEDIAN_STL	0.148	0.436
RT_MEDIAN_SNAP	-.017-	0.929
LT_MEDIAN_SNAP	0.079	0.676
RT_SURAL_STL	.449*	0.013
LT_SURAL_STL	0.096	0.615
RT_SURAL_SNAP	-.503-**	0.005
LT_SURAL_SNAP	-.396-*	0.03

The regression analysis for the significant correlated TSH serum level with electrophysiological parameters of examined nerves were suggestive of TSH serum level which was a significant predictor of affection of the following: 1) conduction velocity of both peroneal and left median nerves. 2) amplitude of compound motor unit action potential of both peroneal nerves and amplitude of sensory nerve action pintail of left sural nerves **(Table 4)**.

Table (4): TSH serum level as a predictor of affection of electrophysiological parameters of examined nerves

Parameters \ Coefficients		Unstandardized Coefficients		Standardized Coefficients	t	sig
		B	Std. Error	BETA		
LT_MEDIAN_CV	(Constant)	13.440	1.174		11.449	<0.001
	TSH	6.733	.200	.975	33.645	<0.001
LT_ULNAR_CV	(Constant)	70.038	8.470		8.269	<0.001
	TSH	-2.251-	1.444	-.201-	-1.559-	0.124
RT_PERONEAL_DML	(Constant)	-1.057-	2.069		-.511-	0.611
	TSH	1.219	.353	.413	3.457	0.001
LT_PERONEAL_DML	(Constant)	5.933	1.838		3.228	0.002
	TSH	0.099	.313	.041	.316	0.753
RT_PERONEAL_CMAP	(Constant)	-2.784-	.479		-5.811-	<0.001
	TSH	0.876	.082	.816	10.731	<0.001
LT_PERONEAL_CMAP	(Constant)	-2.243-	.753		-2.978-	0.004
	TSH	0.755	.128	.611	5.879	<0.001
RT_PERONEAL_CV	(Constant)	-3.759-	7.537		-.499-	0.620
	TSH	8.565	1.285	.659	6.666	<0.001
LT_PERONEAL_CV	5.060	3.507		1.443	.154	5.060
	6.937	0.598	.836	11.604	.000	6.937
RT_SURAL_STL	(Constant)	4.317	.253		17.052	<0.001
	TSH	-0.061-	.043	-.182-	-1.411-	0.164
RT_SURAL_SNAP	(Constant)	5.472	.422		12.970	<0.001
	TSH	0.101	.072	.182	1.411	0.164
LT_SURAL_SNAP	(Constant)	3.229	.408		7.919	<0.001
	TSH	0.510	.070	.694	7.335	<0.001

The risk estimation of neuropathy among the subclinical hypothyroidism and euthyroid groups has shown the subclinical hypothyroidism which was considered as risk factor of neuropathy **(Table 5)**.

Table (5): Risk Estimate

Parameters \ Estimations	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for groups (SCH/ EU)	17.875	5.364	59.568
For cohort diagnosis = neuropathy	8.105	3.034	21.650
For cohort diagnosis = normal	.453	.310	.663
N of Valid Cases	94		

DISCUSSION

In our study, the comparison of age of the subclinical hypothyroidism and the euthyroid groups showed no significant difference in groups matched as age regards. *Karne and Bhalerae (2016)* found that 8% is the prevalence of peripheral neuropathy among the subjects aged ≥55 years, that mean the neuropathy increased as the age increase.

Subclinical hypothyroidism group has a significant difference as regard of CMAP amplitude of both peroneal nerves, conduction velocity of both median, peroneal nerves, sensory terminal latency, SNAP amplitude of both median and sural nerves when compared with euthyroid group. The linear regression analysis confirmed that TSH level is independent predictor of neuropathy among the subclinical hypothyroidism group.

Akarsu et al. (2013) mentioned a changes in electrophysiological measurements as a decrease in conduction velocity and CMAP amplitude and increased motor latency of the ulnar and median nerves. *Balaraman et al (2013)* reported that there is some electrophysiological changes in peripheral nerves of the subclinical hypothyroid females as well as that in early stages of hypothyroid females in comparison with healthy females.

Jaiswal and Dhankad (2020) stated that *Misiunas et al. (1995)* concluded that neuropathy is present in subclinical hypothyroidism, and more evident when basal levels of TSH increased, also report there is electrophysiological measurements alteration in early stages of subclinical hypothyroidism similar to alteration in over myxedema patients, While *Jalilzadh etal (2006)* did not find any abnormality in peripheral nerves of patient with subclinical hypothyroidism.

Gupta, et al 2016 state that *Jalilzadeh et al (2006)* reporting that there is no significant affection of peripheral nerves in subclinical hypothyroidism patients. *Waghmar, et al, (2016)* reported that *Yuskel G et al (2007)* found that electrophysiological abnormalities of the peripheral nerves in subclinical hypothyroidism.

Penza et al. (2009) concluded that subclinical hypothyroidism can cause sensory neuropathy and can be treated with hormonal replacement therapy. *Akarsu et al. (2013)* mentioned a decrease in conduction velocity and CMAP

amplitude and increased motor latency of the ulnar and median nerves.

Waghmar, et al (2016) reported that hormonal and metabolic changes that occur in early stages of hypothyroidism can cause the abnormal electrophysiological measurements of the peripheral nerves. *Jaiswal and Dhankad (2020)* stated that hypothyroidism affect the peripheral nerves and this also to some extent can be seen with cases of subclinical hypothyroidism using neurophysiologic measures. And the hormonal replacement therapy can alleviate the motor bundle and neuromuscular junction affection. and they recommend further studies to conduct clinical evaluation. *Allam et al. (2021)* concluded that the subclinical hypothyroidism is highly prevalent in diabetic polyneuropathy and is independently related to severity. The role of subclinical hypothyroidism in neuropathy can be explained by many mechanisms: The subclinical hypothyroidism increases the oxidative stress, association to pronounced inflammatory state. *Rashdat et al. (2022)* concluded that the severity of clinical symptoms and sings of neuropathy among diabetic patient is related to subclinical hypothyroidism and recommend further studies.

CONCLUSION

The subclinical hypothyroidism has role in peripheral neuropathy as in cases of hypothyroidism so the electrophysiological examination must be done for all cases of subclinical hypothyroidism for early diagnosis of neuropathy and early treatment.

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تاثر العصب الطرفى تحت الاكلينيكى في حالات قصور الغدة الدرقية تحت الإكلينيكي

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الخلفيــة: أحــد أكثــر المســببات شــيوعًا لاعــتلال الأعصــاب الغــدد الصــماء هــو قصور الغدة الدرقية، والذي يمكن أن يسبب اعتلال الأعصاب الطرفية.

الهــدف مــن البحــث: تقيــيم التــأثير اتحــت الاكلينيكــى للعصــب الطرفــى بــين مرضى قصور الغدة الدرقية تحت الإكلينيكي.

المرضـــى وطـــرق البحـــث: تمـــت إحالـــة خمســـين مـــريض مـــن مرضـــى القصـــور الســـريرى للغـــدة الدرقيـــة الـــذين تـــم تشخيصـــهم حـــديثًا مـــن العيـــادة الخارجيـــة لقســـم الباطنيـــة العامـــة إلـــى وحـــدة الفســـيولوجيا العصـــبية فـــي قســـم المـــخ والأعصـــاب بمستشـــفى الأزهـــر الجـــامعى بـــدمياط الجديـــدة ومجموعـــة ضـــابطة مـــن الاصـــحاء مـــن نفـــس اعمـــار المرضـــى وكـــان عـــددهم خمســـون. وتـــم إجـــراء الدراســـة الكهربيـــة لجميـــع المجموعـــات المدروســـة بمـــا فـــي ذلـــك دراســـات التوصـــيل العصـــبي الحركـــي والحســـي لكـــل مـــن الاعصـــاب التالية:الوسيط، والزندي، والشظوي، والربيلى.

نتـــائج البحـــث: كـــان هنـــاك فـــرق ذو دلالـــة احصـــائية بـــين المجمـــوعتين فيمـــا يتعلـــق بســـعة جهـــد العصـــب الحركـــى للعصـــب الشـــظوى، وكـــذلك ســـرعة التوصـــيل للعصـــب الاوســـط ر و الشـــظوي. وكـــان هنـــاك فـــرق ذو دلالـــة احصـــائية بـــين المجمـــوعتين فيمـــا يتعلـــق بـــزمن الكمـــون الطرفـــي الحســـي و ســـعة جهـــد العصـــب الحســـي لكـــلا مـــن العصـــب الوســـط والربيلـــي، و كـــان هنـــاك ارتبـــاط ســـلبي ذو دلالـــة احصـــائية بـــين مســـتوى الهرمـــون المنشـــط للغـــدة الدرقيـــة فـــى الـــدم وكـــلا مـــن ســـرعة التوصـــيل للعصـــب الاوســـط،

والشظوي، و الزندي اليسرى، و سعة جهد العصب الحركى للعصب الشظوى، و سعة جهد العصب الحسي للعصب الربلي الأيمن. وكان هناك ارتباط إيجابي كبير بين مستوى الهرمون المنشط للغدة الدرقية فى الدم و فترة الكمون الحركى لكل من العصب الشظوى بالساقين والعصب الربيلى للساق اليمنى.

الاستنتاج: يمكن اعتبار قصور الغدة الدرقية تحت الإكلينيكي عامل خطر لتأثر الأعصاب الطرفية كما هو الحال في حالات قصور الغدة الدرقية.

الكلمات الدالة: قصور الغدة الدرقية تحت الإكلينيكي، دراسة الفيزيولوجيا الكهربية، اعتلال الأعصاب الغدد الصماء.