MAK HILL Publications Research Journal of Medical Sciences

Research Article
doi: 10.36478/makrjms.2025.2.67.71

A Comparative Study of Eeg Changes in Hypothyroid Individuals and Euthyroid Individuals

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ABSTRACT
Hypothyroidism is a condition characterized by insufficient production of thyroid hormones, which are crucial for metabolic and neurological functions. CNS involvement in hypothyroidism arises due to decreased availability of thyroid hormones, which affects brain metabolism and neuronal signalling. Thyroid hormones regulate myelination, neuronal migration, and synaptogenesis, making hypothyroidism particularly impactful in developmental stages. Common neurological symptoms include fatigue, depression, memory impairment, and difficulty concentration. Severe hypothyroidism may lead to myxoedema, which can cause altered mental status, stupor, or coma in extreme cases. In untreated congenital hypothyroidism, CNS effects may result in intellectual disabilities or delayed developmental milestones. Treatment with levothyroxine typically alleviates CNS symptoms, although recovery may be gradual, depending on the duration of untreated hypothyroidism. Early diagnosis and treatment are critical to prevent irreversible CNS damage and to restore normal neurological function. Thyroid hormone is having direct effect on the electroencephalogram in regard to configuration, frequency and evoked responses. In the previous studies It has been stated that a slowing of dominant rhythm and reduction in amplitude of the EEG background activity, and reduced photic driving can be seen in approximately one-third of hypothyroid patients which may be reversible with treatment. In addition, a decrease in stages 3 and 4 sleeps has been reported in hypothyroidism. To compare the changes of Electroencephalographic activity in hypothyroid individuals and euthyroid individuals. A total of 60 individuals aged 20-50 years participated in this study, divided into two groups as 30 cases and 30 controls. History, demographic details is collected using questionnaires and face-to-face interviews. Anthropometric measures was measured and BMI was calculated. Electroencephalographic recording was conducted in Research Laboratory, Department of Physiology, Tirunelveli medical College, Tirunelveli. After getting written consent from the participants, they were taken to Research laboratory, Department of Physiology between 10 am-1 pm. EEG was recorded in all the individuals and recordings were compared between cases and controls. The association between hypothyroid and EEG waves were found in this study. Results were assessed by using SPSS software 23 version. Using this software mean standard deviation, p values were calculated, unpaired t test was used for analysis, p value less than 0.05 was considered significant. This study concludes that hypothyroidism is significantly correlated with CNS manifestation and highlighting the role of thyroid hormones for normal functioning of brain.

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Key Words
Thyroid hormones, hypothyroid, electro encephalogram, slow wave activity

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Received: 30 November 2024
Accepted: 10 January 2025
Published: 15 January 2025

Citation: R. Vijayalakshmi, M. Anita, H.R. Haribabu and M. Shabiha, 2025. A Comparative Study of Eeg Changes in Hypothyroid Individuals and Euthyroid Individuals. Res. J. Med. Sci., 19: 67-71, doi: 10.36478/makrjms.2025.2.67.71

INTRODUCTION

Thyroid hormone is indispensable for the proper development and functioning of the central nervous system (CNS). Its deficiency, particularly in congenital hypothyroidism, can lead to profound neurological issues. Slowed intellectual functions, including impaired speech, lethargy, memory problems, and reduced cognitive initiative, are common symptoms of thyroid hormone deficiency. These manifestations highlight the critical role thyroid hormones play in maintaining CNS health[1].

Thyroid hormone is particularly critical during early stages of brain development and continues to influence neurological function throughout life. It is involved in key processes such as neuron formation, axonal and dendritic growth, neuronal migration, and synapse formation. Brain regions like the thalamus, cerebellum, hippocampus, caudate nucleus, and cerebral cortex exhibit a higher dependency on thyroid hormone. Additionally, the hormone is essential for the development of the cochlea and retina. By regulating brain-specific genes through distinct receptors, thyroid hormone ensures proper neurological functioning. The timing and regional activity of these receptors play a pivotal role in determining the severity and specificity of deficits caused by thyroid hormone insufficiency[2].

Hypothyroidism is one of the most common endocrine disorders, affecting over 1% of the general population and nearly 5% of individuals above 60 years of age.[3]It is a syndrome characterised by the clinical and biochemical manifestation of thyroid hormone deficiency in the target tissue[4].

Iodine deficiency remains the most common cause of hypothyroidism worldwide. In areas of iodine sufficiency, autoimmune disease (Hashimotos thyroiditis) and Iatrogenic causes (treatment for hypothyroidism) are most common[5].

Metabolic abnormalities associated with hypothyroidism, including reduced cerebral blood flow and the accumulation of mucopolysaccharides, are thought to contribute to the neurological symptoms observed in these patients. These findings highlight the essential role of thyroid hormone in both the developmental and ongoing functionality of the CNS[6].

Electroencephalography (EEG) is a diagnostic tool that records electrical activity in the brain, often used to evaluate neurological conditions. In the context of hypothyroidism, EEG patterns can provide insights into the impact of thyroid hormone deficiency on brain function. Hypothyroidism is known to alter normal brain activity, which is often reflected as abnormalities in EEG readings. Research suggests that thyroid hormone deficiency affects neuronal excitability and synaptic transmission, leading to these EEG abnormalities. These findings reinforce the critical role of thyroid hormone in maintaining normal brain electrophysiology and emphasize the utility of EEG in identifying and monitoring neurological impairments associated with hypothyroidism. This study aims to correlate the EEG findings between Euthyroid and Hypothyroid individuals.

MATERIALS AND METHODS

This cross-sectional study involves a total of 60 participants, calculated using Open Epi software, divided into two groups: Group A (cases) with 30 hypothyroid individuals and Group B (controls) with 30 normal individuals.

Inclusion criteria include participants aged 20–50 years, both males and females, with hypothyroidism (TSH > 5.1 mIU/L), and those willing to participate. Exclusion criteria rule out individuals with cardiorespiratory diseases, physical disabilities, seizure disorders, diabetes mellitus, hypertension, alcohol or smoking habits, medications affecting EEG, prior head injuries, or psychiatric disorders.

Electroencephalography (EEG) will measure the brain's spontaneous electrical activity, generated by summated postsynaptic potentials beneath the scalp electrodes. EEG recordings were performed for all participants and compared between the groups. Data collection includes gathering history and demographic details through questionnaires and interviews, measuring anthropometric parameters to calculate BMI, and conducting EEG recordings in the Research Laboratory, Department of Physiology, Tirunelveli Medical College, Tirunelveli. Written consent was obtained, and participants were tested between 10 AM and 1 PM.

Quality assurance involves recording all data, including history, anthropometric measurements, and biochemical investigations, in a master Excel sheet. Blood pressure will be measured using a mercury sphygmomanometer, and EEG was recorded with standardized protocols.

Electroencephalography (EEG) is a non-invasive procedure used to record brain activity, and proper preparation is essential for accurate results. Before the procedure, patients were informed that EEG is painless and safe. The patient is advised to avoid fasting to prevent hypoglycaemia. They were advised avoid caffeine, alcohol, and stimulants for 8–12 hours before the test and wash their hair the night before without applying conditioner or spray. The patient should be positioned comfortably in a reclining chair and the EEG was recorded in a quiet room. During the procedure, electrodes are placed on the scalp following the International 10-20 system, with the scalp cleaned and conductive gel applied for better signal quality. Throughout the procedure, any artifacts caused by movement, blinking, or muscle activity are monitored

and addressed. EEG was recorded using RMS Acquisition and analysis software. After the recording, electrodes are removed, and the scalp was cleaned. The patient was then debriefed about the process and any observations. This systematic approach ensures accurate and reliable EEG results.

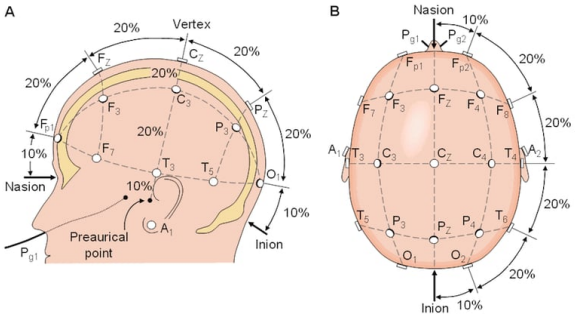

Fig 1: The 10-20 system with front-back (nasion to inion) 10% and 20% electrode distances

RESULTS AND DISCUSSIONS

Table 1 ensures that the cases and controls are matched for age, height, and weight. This matching minimizes the potential confounding effects of these variables, ensuring that the study results accurately reflect the impact of hypothyroidism on EEG parameters, confirming that these demographic and physical factors do not influence the observed differences in EEG wave characteristics."

This study shows, the peak latencies of various waves in the EEG are often delayed when compared to age- and sex-matched healthy individuals. This delay indicates a slower neural conduction, which is a common feature of hypothyroidism. The alpha frequency and amplitude were notably reduced in cases compared to controls, with the difference being statistically significant ($p < 0.05$). Additionally, there was an increased presence of slow wave activity (delta waves) in cases when compared to controls, highlighting a distinct alteration in EEG patterns associated with hypothyroidism.

Hypothyroid individuals commonly exhibit an increase in slow wave activity on EEG, particularly in the delta and theta frequency bands. This finding is associated with the generalized slowing of brain processes and may correlate with symptoms such as cognitive dysfunction or fatigue that are typical in hypothyroidism.

Table 1: Comparision of Age, BMI Between Cases and Controls

	Cases/ controls	Mean	Std. Deviation	Mean ± Std.
Age	Case	33.47	11.57	33.47± 11.57
	Controls	33.47	11.57	33.47± 11.57
Ht(cm)	Case	160.57	6.7	160.57± 6.7
	Controls	160.17	6.79	160.17± 6.79
Wt(kg)	Case	56.03	10.28	56.03 ± 10.28
	Controls	56.37	9.58	56.37 ± 9.58
BMI	Case	21.96	3.98	21.96 ± 3.98
	Controls	21.96	3.98	21.96 ± 3.98

Table 2: Comparision of TSH Between Cases and Controls

	Case/ Controls	Mean	Std. Deviation	Mean ± Std.
TSH(mIU/L)	Case	7.18	1.13	7.18 ± 1.13
	Controls	3.36	1.09	3.36 ± 1.09

The prevalence of subclinical hypothyroidism is reported to be between 4% and 10%[7]. Hypothyroidism significantly impacts neurological functions due to the deficiency of thyroid hormones, which are crucial for maintaining neuronal excitability, synaptic transmission, and myelination. The prolonged peak latencies observed in hypothyroid individuals can be attributed to slowed nerve conduction resulting from demyelination and impaired axonal transport. Additionally, reduced synaptic efficiency due to altered neurotransmitter release and uptake further contributes to delayed neural transmission. Cerebral hypoperfusion in hypothyroidism exacerbates these delays by impairing blood flow to critical brain regions. The smaller amplitudes in hypothyroid patients reflect reduced cortical excitability, stemming from disrupted ion channel regulation and neurotransmitter sensitivity. Chronic hypothyroidism can also induce neuroinflammation, impairing normal neuronal firing patterns and synaptic plasticity. These changes result in diminished neural responsiveness and lower recorded amplitudes during neurophysiological assessments.

An increase in slow-wave activity, particularly delta and theta waves, is another hallmark of hypothyroidism, reflecting generalized slowing of EEG patterns. This is caused by metabolic depression in the brain, which operates at a lower metabolic rate in the absence of sufficient thyroid hormones. Disruption of thalamocortical networks, responsible for regulating wakefulness and cognitive processing, further promotes the dominance of slow-wave rhythms.

Clinically, these findings correlate with common symptoms of hypothyroidism, such as cognitive impairment, memory deficits, and sluggish thinking. Motor slowness, including bradykinesia and fatigue, aligns with prolonged latencies and reduced amplitudes observed in neurophysiological studies. Fatigue and lethargy in hypothyroid individuals are mirrored by the increased slow-wave activity detected on EEG.

Hypothyroidism is associated with central nervous system (CNS) impairment due to its influence on brain metabolism and neuronal activity. Studies have shown that reduced thyroid hormone levels result in alterations in neurotransmitter function and cerebral blood flow, leading to cognitive dysfunction and electrophysiological changes[8].

Electroencephalographic studies indicate a predominance of slow-wave activity in hypothyroid patients, suggesting delayed cortical processing and reduced neuronal excitability[9].

Table 3: Comparision of Alpha Frequency and Amplitude Between Cases and Controls

		Mean(sd)	Median	Mean Difference	p-value
α Frequency (8-13Hz)	Cases	8.42(1.70)	8.25	1.17	0.003*
	controls	9.58(1.21)	10		
Amplitude	Cases	61.60(16.12)	60	13.8	<0.001*
	controls	75.40(9.58)	75		

*statistically significant. p value <0.05

Table 4: Comparision of Slow Wave Activity Between Cases and Controls

D	Cases	Controls	Odds Ratio (95% CI)	P Value
Present	9(30%)	0	27(1.49 to 488)	0.001*
Absent	21(70%)	30(100%)		

*statistically significant. p value <0.05

A decrease in the amplitude of alpha, beta, and theta waves has been observed, reflecting impaired synaptic function and reduced cortical responsiveness[10].

These changes are attributed to the direct impact of thyroid hormones on neuronal membrane stability and ion channel function, further underscoring the neurophysiological disruptions caused by hypothyroidism[11].

The reduction in wave amplitude, particularly in alpha and beta bands, has been linked to slowed information processing and reduced attention spans in hypothyroid individuals[12].

Moreover, the presence of diffuse slow-wave patterns correlates with symptoms such as lethargy and decreased alertness, highlighting the systemic impact of thyroid hormone deficiency on CNS functioning[13].

EEG abnormalities in hypothyroidism are associated with cognitive dysfunction, particularly slowed information processing[14,15] discusses EEG recordings during thyroid hormone deprivation and replacement in patients with minimal functional thyroid tissue.

Heinrich[16] reports generalized slow wave abnormalities and sporadic epileptiform abnormalities in patients with Hashimoto encephalitis, a condition associated with hypothyroidism.

This condition is more common among the elderly, with a higher prevalence in females compared to males[17]. Hypothyroidism often impacts the central nervous system, leading to delays in neural conduction. These include abnormalities such as delayed latencies in auditory brainstem evoked potentials and visual evoked potentials, as well as reduced amplitudes [18,19].

The most frequently observed EEG abnormality was diffuse slowing of the background activity[20,21].

Thyroxin deficiency has a significant effect on brain function, with early and notable symptoms including visual impairment and slower cognitive processing[22].

During the assessment of pregnant women with diffuse toxic goiter, EEG recordings revealed patterns of slow-wave and acute-wave activity. These were accompanied by signs of irritation in the medio-basal brain structures, such as bursts of sharp alpha-wave activity[23]. Hypothyroidism is known to severely affect cerebral metabolism, particularly in children compared to adults[24-27].

A slowing of dominant rhythm and a reduction in amplitude of the EEG background activity3 can be seen in approximately one-third of hypothyroid patients' which may be reversible with treatments. In addition, a decrease in stages 3 and 4 of sleep has been reported in hypothyroidism[28].

These findings collectively indicate a significant role of hypothyroidism in altering brain function, as evidenced by electrophysiological assessments like EEG.

CONCLUSION

The above observations emphasize the importance of early detection and treatment of hypothyroidism to prevent long-term neurological impairments. Regular neurophysiological testing, including EEG and event-related potentials, can aid in monitoring disease progression and therapy effectiveness. Understanding these neurological impacts provides a deeper insight into the systemic nature of hypothyroidism, underscoring its influence beyond metabolic functions.

Limitations: The present study has several limitations that need to be acknowledged. First, the sample size of 60 subjects (30 cases and 30 controls) is relatively small, which may limit the generalizability of the findings. Variations in the severity of hypothyroidism among the cases were not analyzed, which might have impacted the results. The use of the 10-20 EEG system, though standard, may have missed subtle or localized brain activity differences. Lastly, the cross-sectional design of the study provides only a snapshot of EEG changes, and a longitudinal approach could offer deeper insights into the progression of these changes in hypothyroidism.

Funding: None of the authors received funding for this study.

Competing Interest: There is no competing interest

Authors Contribution: All authors in our study contributed to the data collection of the patients

Acknowledgement: The authors like to thank the Dean of the Medical College,Head of the Department of Physiology, Tirunelveli Medical College, Tirunelveli,TamilNadu.

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