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Original Research Article

ESTIMATION OF THYROID PROFILE IN ADULT PATIENTS WITH EPILEPSY ON ANTIEPILEPTIC DRUGS ATTENDING THE OUT-PATIENT DEPARTMENT IN A TERTIARY CARE HOSPITAL – A CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Epilepsy is a chronic neurological disorder requiring long-term antiepileptic drug therapy. Prolonged use has been linked to endocrine thyroid dysfunction. Subclinical hypothyroidism, defined by elevated thyroid-stimulating hormone with normal thyroid hormone levels, is often asymptomatic and underdiagnosed. The objective is to estimate serum thyroid hormone levels, determine the prevalence of subclinical and overt thyroid dysfunction, and assess associations with individual antiepileptic drugs, duration of therapy, and treatment regimen. **Materials and Methods:** This cross-sectional observational study was conducted over twelve-months in the outpatient department of medicine at a tertiary care hospital. Adult patients more than 18 years with epilepsy receiving antiepileptic drug therapy for at least six months were included. Serum T3, T4, and TSH levels were measured using chemiluminescent immunoassay. Data were analysed using descriptive statistics and Chi-square or Fisher's exact test, with $p < 0.05$ considered statistically significant. **Result:** A total of 77 patients were included in the study, comprising 44 males (57.1%) and 33 females (42.9%). Subclinical hypothyroidism was identified in 28 patients (36.4%). Thyroid dysfunction showed no significant association with age, sex, individual antiepileptic drugs, or monotherapy versus polytherapy ($p > 0.05$). However, a significant association was observed between duration of antiepileptic drug therapy and subclinical hypothyroidism, with higher prevalence among patients treated for more than five years ($p = 0.009$). **Conclusion:** Subclinical hypothyroidism is common among patients with epilepsy receiving long-term AED therapy. Longer duration of treatment significantly increases the risk, highlighting the importance of periodic thyroid function monitoring in patients on prolonged antiepileptic treatment.

INTRODUCTION

Epilepsy is a persistent neurological condition marked by repeated, unprovoked seizure episodes and impacts nearly 50 million individuals worldwide, ranking among the most prevalent neurological disorders globally.[1] Antiepileptic drugs (AEDs) remain the cornerstone of epilepsy management; however, commonly prescribed agents such as phenytoin, sodium valproate, and carbamazepine exhibit marked inter-individual variability in bioavailability, which may result in therapeutic failure or drug-related toxicity. Genetic and epigenetic factors have been proposed as important contributors to this variability.[2]

Subclinical hypothyroidism is defined biochemically by elevated serum thyroid-stimulating hormone (TSH) levels ranging from 4 to 10 mIU/L, with normal serum free thyroxine (FT4) and free triiodothyronine (FT3) concentrations; the normal reference range for TSH is 0.4–4 mIU/L.[2,3] Desai et al. reported a prevalence of subclinical hypothyroidism of 32% in the Indian population.[4] Other epidemiological studies from India have documented prevalence rates ranging from 9% to 26%.[3]

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Several studies have demonstrated that long-term use of antiepileptic drugs is associated with hormonal, metabolic, and vascular disturbances.[2] Subclinical hypothyroidism poses a diagnostic challenge because classical clinical features of hypothyroidism are often absent.[2,5] Previous studies by Lai et al. and Larkin et al. have demonstrated an association between long-term antiepileptic drug therapy and reduced serum thyroid hormone levels.[2,6,7] Despite the widespread and prolonged use of antiepileptic drugs, routine monitoring of thyroid function is not universally practiced. Furthermore, there is limited Indian data evaluating thyroid profile abnormalities in adult patients receiving long-term AED therapy. Hence, the present study aims to estimate serum levels of thyroid hormones in adult patients with epilepsy on antiepileptic drug therapy, to determine the prevalence of subclinical hypothyroidism in these patients and to evaluate the relationship between duration of antiepileptic drug therapy and thyroid dysfunction.

MATERIALS AND METHODS

Patient population and Ethical consideration

The present a cross-sectional observational study was conducted in the Outpatient Department of Medicine at a tertiary care hospital. Adult patients diagnosed with epilepsy and receiving AED therapy who attended the outpatient department during the study period of 12 months were the study population. The sample size was calculated based on the prevalence of subclinical hypothyroidism among patients receiving antiepileptic drugs reported in previous studies. Due to feasibility and time constraints, a convenient sample size of 77 participants was included. The study was initiated after obtaining approval from the Institutional Human Ethics Committee (Ref No: 1748/ME3/2024). Written informed consent was obtained from all participants in the local vernacular language before their inclusion in the study. Confidentiality was maintained throughout the study.

Selection criteria: Adult patients aged 18 years and above of both genders, diagnosed cases of epilepsy receiving antiepileptic drug therapy for at least six months, were included in the study. Patients with known thyroid disorders before initiation of AED therapy, Patients receiving thyroid hormone replacement or anti-thyroid medications, pregnant or lactating women, patients with chronic systemic illnesses such as chronic liver disease or chronic kidney disease and patients receiving drugs known to affect thyroid function (e.g., amiodarone, lithium) were excluded from the study.

Study procedure: Eligible participants were enrolled as per the selection criteria. Demographic details such as age and sex were recorded. A detailed clinical history, including duration of epilepsy, type of antiepileptic drug (monotherapy or polytherapy), dosage, duration of treatment, and concomitant medications, was documented using a pre-designed case record form. A thorough physical and systemic examination was carried out, and parameters including height, weight, body mass index, heart rate, and blood pressure were recorded. After an overnight fasting period of 12 hours, 5 mL of venous blood was collected under aseptic precautions. Serum levels of triiodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH) were estimated using standard laboratory methods such as chemiluminescent immunoassay. In addition, liver function tests were performed. Laboratory reports were recorded for analysis. Subclinical hypothyroidism refers to a state in which serum thyroid-stimulating hormone levels are elevated (4–10 mIU/L) while serum free thyroxine and free triiodothyronine concentrations remain within the normal range, without clinical manifestations of overt hypothyroidism.[2,3]

Statistical Analysis: Data were entered into Microsoft Excel and analysed using SPSS software. Descriptive statistics were used to summarise demographic and clinical variables. Categorical variables were expressed as frequencies and percentages, while continuous variables were expressed as mean ± standard deviation. The association between antiepileptic drugs and thyroid dysfunction was analysed using the Chi-square test or Fisher's exact test as appropriate. A p-value of < 0.05 was considered statistically significant.

RESULTS

The study included a total of 77 patients diagnosed with epilepsy. Of the total participants, 44 were males (57.1%) and 33 were females (42.9%), indicating a higher male representation. The most common age group was ≤30 years (n = 22; 32.5%), followed by 41–50 years (n = 16; 20.8%). Patients aged 31–40 years (n = 13; 16.9%) and >60 years (n = 13; 16.9%) were equally represented. Nearly half of the participants were employed (n = 39; 50.6%). Comorbid conditions were present in 29 (37.7%) patients. Generalized epilepsy was the most frequent seizure type (n = 40; 51.9%), followed by focal epilepsy (n = 34; 44.2%). With respect to duration of antiepileptic treatment, the majority of patients had been receiving treatment for 1–5 years (n = 41; 53.2%), while 28 (36.4%) patients had been on treatment for more than five years. Polytherapy was prescribed to 47 (61.0%) patients, whereas monotherapy was used in 30 (39.0%) patients. Most participants were non-vegetarians (n = 76; 98.7%). A history of smoking was reported by 14 (18.2%) patients, and a family history of epilepsy was present in 7 (9.1%) patients [Table 1].

Among the 77 study participants, subclinical hypothyroidism was observed in 28 (36.4%) individuals, while the remaining 49 (63.6%) participants were euthyroid with no evidence of subclinical hypothyroidism. [Table 2] The mean total

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T3 level among the participants was 0.84 ± 0.66 ng/ml, with values ranging from 0.1 to 2.9 ng/ml. The mean serum T4 level was 7.83 ± 3.40 µg/dl, while the mean TSH was 4.64 ± 3.49 mIU/L, showing a wide variability across the study population. Liver enzyme analysis showed a mean alanine aminotransferase level of 32.3 ± 13.6 U/L and a mean aspartate aminotransferase level of 29.8 ± 11.1 U/L, with both parameters demonstrating ranges extending beyond typical upper limits in some participants. [Table 3] Subclinical hypothyroidism was observed across all age groups. A relatively higher proportion was noted among participants aged 31–40 years (n = 6; 46.2%), 51–60 years (n = 5; 50.0%), and >60 years (n = 7; 53.8%). Lower proportions were noted in the ≤30 years (n = 6; 24.0%) and 41–50 years (n = 4; 25.0%) age groups. Although the proportion of subclinical hypothyroidism appeared to increase with advancing age, the association between age group and presence of subclinical hypothyroidism was not statistically significant (Pearson's χ^2 test; p = 0.231) [Table 4].

Table 1: Baseline characteristics (n = 77)

Parameters	Frequency (n)	Percentage (%)
Gender		
Male	44	57.1
Female	33	42.9
Age group (years)		
≤ 30	25	32.5
31–40	13	16.9
41–50	16	20.8
51–60	10	13.0
> 60	13	16.9
Occupation		
Employed	39	50.6
Unemployed	38	49.4
Personal History		
Diet		
Vegetarian	1	1.3
Non-vegetarian	76	98.7
Smoking history	14	18.2
Family history of epilepsy	7	9.1
Comorbid condition		
Yes	29	37.7
No	48	62.3
Type of epilepsy		
Focal	34	44.2
Generalized	40	51.9
Combined	1	1.3
Unknown	2	2.6
Duration of treatment		
< 1 year	8	10.4
1–5 years	41	53.2
> 5 years	28	36.4
AED regimen		
Monotherapy	30	39.0
Polytherapy	47	61.0

Table 2: Prevalence of subclinical hypothyroidism (N = 77)

Thyroid status	n	%
Subclinical hypothyroidism	28	36.4
Euthyroid (no subclinical hypothyroidism)	49	63.6

Table 3. Descriptive statistics of biochemical parameters (N = 77)

Variable	Mean	SD	Min	Max
Total T3 (ng/ml)	0.84	0.66	0.1	2.9
T4 (µg/dl)	7.83	3.40	3.5	24.9
TSH (mIU/L)	4.64	3.49	0.0	14.4
ALT (U/L)	32.3	13.6	15.0	71.0
AST (U/L)	29.8	11.1	15.0	68.0

Table 4: Association between age group and subclinical hypothyroidism

Age group (years)	Subclinical hypothyroidism n (%)	No subclinical hypothyroidism n (%)	Total n	p value (Pearson χ^2)
≤ 30	6 (24.0)	19 (76.0)	25	0.231
31–40	6 (46.2)	7 (53.8)	13	
41–50	4 (25.0)	12 (75.0)	16	
51–60	5 (50.0)	5 (50.0)	10	
> 60	7 (53.8)	6 (46.2)	13	
Total	28 (36.4)	49 (63.6)	77	

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Subclinical hypothyroidism was observed among patients receiving various antiepileptic drugs, with phenytoin being the most commonly used drug; 19 (32.8%) patients on phenytoin had subclinical hypothyroidism. In patients receiving sodium valproate, subclinical hypothyroidism was present in 12 (37.5%) patients, comparable proportions were noted with 12 (37.5%) patients receiving sodium valproate, 4 (40.0%) patients receiving carbamazepine, 5 (33.3%) patients receiving levetiracetam, and 5 (45.5%) patients receiving clonazepam. No cases of subclinical hypothyroidism were observed among patients receiving oxcarbazepine or phenobarbitone, though the numbers in these groups were very minimal. Fisher's exact test did not demonstrate a statistically significant relationship between any individual antiepileptic drug and the development of subclinical hypothyroidism (all p values > 0.05). This suggests that, in this study population, no single AED was independently associated with subclinical hypothyroidism. [Table 5]

Table 5: Association of antiepileptic drugs and subclinical hypothyroidism

Antiepileptic drug	Subclinical hypothyroidism n (%)	No subclinical hypothyroidism n (%)	Fisher exact test p value
Phenytoin	19 (32.8)	39 (67.2)	0.281
Sodium valproate	12 (37.5)	20 (62.5)	1.000
Diazepam	3 (37.5)	5 (62.5)	1.000
Clonazepam	5 (45.5)	6 (54.5)	0.737
Carbamazepine	4 (40.0)	6 (60.0)	1.000
Levetiracetam	5 (33.3)	10 (66.7)	1.000
Oxcarbazepine	0 (0.0)	1 (100.0)	1.000
Phenobarbitone	0 (0.0)	2 (100.0)	0.531

The prevalence of subclinical hypothyroidism varied with the duration of antiepileptic drug treatment. Among patients treated for less than one-year, subclinical hypothyroidism was observed in 3 (37.5%) patients. A lower proportion was noted among those treated for 1–5 years (n = 9; 22.0%). In contrast, a higher proportion was observed among patients receiving treatment for more than five years, where 16 (57.1%) patients were affected. The association between duration of antiepileptic drug treatment and subclinical hypothyroidism was statistically significant (Pearson's χ^2 test; p = 0.009), indicating that that prolonged antiepileptic drug therapy is significantly associated with an increased risk of subclinical hypothyroidism [Table 6].

Table 6: Association between duration of treatment with antiepileptic drugs and subclinical hypothyroidism

Variable	Category	Subclinical hypothyroidism n (%)	No subclinical hypothyroidism n (%)	Chi-square test p value
Duration of treatment	< 1 year	3 (37.5)	5 (62.5)	0.009*
	1–5 years	9 (22.0)	32 (78.0)	
	> 5 years	16 (57.1)	12 (42.9)	

Comparison between monotherapy and polytherapy revealed no statistically significant differences in thyroid function, as the observed mean differences were small and all confidence intervals included zero [Table 7].

Table 7: Differences in thyroid function between patients on monotherapy and polytherapy

Parameter	Group	N	Mean ± SD	Mean difference (95% CI)	p-value
Total T3 (ng/ml)	Monotherapy	30	0.716 ± 0.483	-0.195 (-0.476 to 0.085)	0.169
	Polytherapy	47	0.912 ± 0.752		
T4 (µg/dl)	Monotherapy	30	7.703 ± 1.960	-0.199 (-1.585 to 1.188)	0.776
	Polytherapy	47	7.902 ± 4.088		
TSH (mIU/L)	Monotherapy	30	4.638 ± 3.505	-0.009 (-1.647 to 1.630)	0.992
	Polytherapy	47	4.647 ± 3.511		

DISCUSSION

The present cross-sectional study evaluated the prevalence of subclinical hypothyroidism and its association with demographic factors, antiepileptic drug use, and treatment-related variables among patients with epilepsy attending a tertiary care hospital. Subclinical hypothyroidism was observed in 36.4% of the study population, indicating a relatively high burden of thyroid dysfunction in patients receiving long-term AED therapy. The overall prevalence of subclinical hypothyroidism in this study (36.4%) is higher than that reported in the general population, suggesting a possible influence of chronic AED exposure. The prevalence of subclinical hypothyroidism has been reported to range from 3% to 15%, depending on the study population and diagnostic criteria used. Epidemiological studies consistently demonstrate a higher incidence among women and older individuals, indicating the effect of

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age and sex on thyroid dysfunction. [8-10] These findings emphasise the importance of thyroid function surveillance in epilepsy patients, especially in those receiving prolonged treatment.

In the baseline characteristics, males constituted a higher proportion of participants, which is comparable to several Indian hospital-based epilepsy studies where male predominance has been attributed to higher health-care-seeking behaviour and referral patterns. The majority of patients were younger than 40 years, reflecting the common occurrence of epilepsy in the economically productive age group. Generalised epilepsy was the most frequent seizure type, followed by focal epilepsy, consistent with earlier epidemiological observations from tertiary care centres. In India, epilepsy prevalence is slightly higher among males due to greater exposure to risk factors such as head trauma and alcohol use.[11] Globally, females have a marginally lower annual incidence of epilepsy than males, as reported by Kotsopoulos et al.[12] The majority of patients with epilepsy are younger than 40 years of age, the global data show the highest number of prevalent epilepsy cases occurring in the 15 to 49 year age group.[13] Generalised epilepsy was the most frequent seizure type observed, similar to findings reported in other tertiary care epilepsy cohorts.[14]

Subclinical hypothyroidism was observed across all age groups, with relatively higher proportions among patients aged above 30 years, particularly those over 50 years. Although an increasing trend with age was evident, the association did not reach statistical significance. This observation parallels an earlier study suggesting that age may act as a contributory but not independent risk factor for thyroid dysfunction in patients on antiepileptic drugs, where treatment-related factors such as polytherapy and enzyme-inducing medications appear to play a more dominant role. Previous research has shown that changes in thyroid hormone levels in epilepsy patients on long-term AED therapy are associated with multiple variables, including drug type and duration of treatment, rather than age alone, and that the mechanisms often involve altered hormone metabolism and hypothalamic-pituitary-thyroid axis interference due to the drugs themselves.[15]

Analysis based on individual AEDs did not reveal a statistically significant association between any single drug and the presence of subclinical hypothyroidism. While relatively higher proportions were observed with phenytoin, sodium valproate, carbamazepine, and clonazepam, these differences were not significant. Previous studies have reported variable effects of individual AEDs on thyroid function, often influenced by duration of therapy, drug combinations, and study design.[15] The frequent use of polytherapy in the present study may have further limited the ability to isolate drug-specific effects.

A key finding of this study was the significant association between duration of AED therapy and subclinical hypothyroidism. A significantly higher prevalence of thyroid dysfunction was observed among patients receiving antiepileptic drugs for more than five years compared with those treated for shorter periods. This supports the hypothesis of a cumulative effect of prolonged AED exposure on thyroid function. Similar duration-dependent associations have been reported in both adult and paediatric populations and long-term enzyme induction, altered hormone binding, and changes in hypothalamic–pituitary–thyroid axis regulation have been proposed as possible mechanisms. Chronic antiepileptic therapy has been linked to alterations in thyroid hormone metabolism and homeostasis, with mechanisms including enhanced hepatic enzyme induction, increased hormone degradation and modifications in protein binding mechanisms or impairment of the hypothalamic–pituitary–thyroid axis function. [16,17]

Comparison between monotherapy and polytherapy groups did not demonstrate significant differences in serum T3, T4, or TSH levels. Although polytherapy patients had marginally higher mean hormone levels, the differences were clinically insignificant. Similar findings have been reported by previous studies, suggesting that thyroid hormone changes over time during AED therapy, noting changes with prolonged exposure. Twelve-month observational study of children on various AEDs reported thyroid hormone changes during prolonged treatment, highlighting effects over time irrespective of monotherapy vs. polytherapy.[18]

A previous study found lower thyroid hormone levels in polytherapy patients compared with monotherapy patients, but the differences were not statistically significant, indicating comparable thyroid profiles irrespective of monotherapy or polytherapy.[19]

The clinical implications of these findings are important. Subclinical hypothyroidism, though often asymptomatic, has been associated with adverse cardiovascular outcomes, neurocognitive impairment, and reduced quality of life.[20] Evidence shows that higher TSH levels in subclinical hypothyroidism are linked with increased CHD risk in patients with epilepsy; unrecognised thyroid dysfunction may further contribute to fatigue, cognitive slowing, and poor seizure control. Accordingly, regular assessment of thyroid function may be justified, especially in patients receiving long-term antiepileptic drug therapy.[16]

This study has certain limitations. The cross-sectional design does not allow determination of a causal association between antiepileptic drug exposure and subclinical hypothyroidism. As the study was carried out at a tertiary care centre with a limited sample size, the results may not be generalisable. Many patients were on polytherapy, limiting the assessment of individual drug effects. Baseline thyroid function was unavailable, so pre-existing dysfunction cannot be ruled out, especially in older patients or those with comorbidities. Despite these limitations, the study provides valuable insight into subclinical hypothyroidism in epilepsy and supports regular

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thyroid monitoring in patients on long-term AED therapy. Larger longitudinal studies are needed to clarify causal relationships and drug-specific effects. Strengths of the Study is that, this study provides important data on the prevalence of subclinical hypothyroidism among epilepsy patients in a tertiary care setting, assessment of serum T3, T4, and TSH enabled accurate identification of subclinical hypothyroidism, analysis of age, epilepsy type, AED duration, individual drugs, and treatment regimen identified treatment duration as a key determinant of thyroid dysfunction and finally, appropriate statistical tests and reporting of confidence intervals strengthened result validity. The findings of the study highlight an under-recognised adverse effect of long-term AED therapy and support routine thyroid function monitoring in epilepsy patients.

CONCLUSION

In conclusion, subclinical hypothyroidism was common among patients with epilepsy receiving AED therapy, particularly in those on long-term treatment. While no individual AED was independently associated with thyroid dysfunction, longer duration of treatment showed a significant association. Routine thyroid function monitoring may be considered in patients receiving prolonged antiepileptic treatment.

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