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Original Research Article

EFFECT OF ANTICONVULSANT MONOTHERAPY ON LIPID PROFILE IN CHILDREN: A CASE–CONTROL STUDY

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ABSTRACT

Background: Antiepileptic drugs (AEDs) are essential for seizure control in children but may alter lipid metabolism, potentially increasing long-term cardiovascular risk. Evidence regarding lipid profile changes in pediatric patients receiving AED monotherapy remains limited.

Materials and Methods: A hospital-based case–control study was conducted among children (<12 years) receiving single-drug AED therapy for at least six months. Thirty-eight cases and 38 age- and sex-matched healthy controls were included. Fasting serum lipid parameters—total cholesterol, LDL-C, HDL-C, triglycerides, and VLDL-C—were analyzed using standard enzymatic methods. Statistical analysis was performed using SPSS, with $p < 0.05$ considered significant.

Results: Children receiving phenytoin showed significantly higher total cholesterol, LDL-C, and triglyceride levels and lower HDL-C compared to controls ($p < 0.001$). No significant lipid alterations were observed in children treated with levetiracetam or sodium valproate. Demographic factors and duration of epilepsy did not influence lipid outcomes, while a positive family history of epilepsy was more common among cases.

Conclusion: Phenytoin therapy was associated with adverse lipid profile changes in children, whereas levetiracetam and sodium valproate had minimal metabolic effects. Lipid monitoring is advisable in pediatric patients receiving enzyme-inducing AEDs.

Keywords: Epilepsy, Children, Antiepileptic drugs, Phenytoin, Lipid profile, Dyslipidemia.

INTRODUCTION

Epilepsy is one of the most common chronic neurological disorders, affecting nearly 50 million people worldwide, with a higher burden in low- and middle-income countries.[1] It represents a major cause of neurological morbidity in children and frequently requires long-term treatment with antiepileptic drugs (AEDs).[2] Although these medications are essential for seizure control, they are known to exert systemic effects, including alterations in lipid metabolism.[3] This association is of particular clinical concern in pediatric patients, as early metabolic disturbances may predispose them to future cardiovascular complications.[4]

The lipid profile — consisting of serum total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides — is a key indicator of cardiovascular health.[5] Dyslipidemia, defined by abnormalities in these lipid fractions, plays a central role in the development of atherosclerosis and metabolic syndrome.[6] While the effects of AEDs on lipid metabolism have been

extensively studied in adults, data in the pediatric population remain limited.[7] Children receiving long-term AED therapy constitute a vulnerable group, as ongoing growth, developmental metabolic demands, and prolonged drug exposure may enhance susceptibility to metabolic adverse effects.[8]

The impact of AEDs on lipid metabolism is largely attributed to their influence on hepatic enzyme systems, particularly cytochrome P450 enzymes.[9] Conventional AEDs such as phenobarbital, phenytoin, and carbamazepine are potent enzyme inducers and may accelerate lipid metabolism, resulting in altered serum lipid concentrations.[10] In contrast, newer AEDs such as levetiracetam and lamotrigine are non-enzyme inducers and demonstrate a different metabolic profile.[11] This pharmacological distinction underscores the need to evaluate their differential effects on lipid parameters.[12]

Several studies have reported an association between long-term AED therapy and dyslipidemia in children. Verrotti et al. observed significantly elevated total cholesterol and LDL levels in children receiving enzyme-inducing AEDs compared to healthy controls.[13] Similarly, Bhadran et al. documented comparable metabolic alterations in South Indian children, particularly those treated with carbamazepine and valproate.[14] However, the influence of newer AEDs on lipid metabolism remains inconclusive, with some studies suggesting minimal or no significant changes.[15] Given these inconsistencies and the limited pediatric data, further evaluation is warranted.

Objectives

- To evaluate the effect of single-drug anticonvulsant (antiepileptic) therapy on serum lipid profile (total cholesterol, LDL, and HDL) in children with seizure disorders.
- To determine the frequency of lipid profile abnormalities among children receiving commonly prescribed anticonvulsant monotherapy.

MATERIALS AND METHODS

This case–control study was conducted at a tertiary care hospital located at Visakhapatnam. The study was carried out in the pediatric outpatient and inpatient departments over a period of 18 months. The study population comprised two groups: cases and controls. The case group included children with seizure disorders who had been receiving single-drug antiepileptic therapy (AED monotherapy) for a minimum duration of six months and were under regular follow-up at the hospital. The control group consisted of apparently healthy children who were age- and sex-matched with the cases.

Children aged less than 12 years who had been on single-drug AED therapy for at least six months were included in the study. Children with conditions known to affect lipid metabolism, such as nephrotic syndrome, thyroid disorders, other endocrinopathies, chronic liver, cardiac, or renal diseases, and progressive neurological or psychiatric illnesses were excluded. Those receiving combination AED therapy and children whose guardians did not provide informed consent were also excluded.

After obtaining written informed consent from parents or guardians, a detailed clinical evaluation was performed for each participant to confirm eligibility. Demographic and clinical information including age, sex, type of seizures, duration of AED therapy, specific drug used, and family history of seizures were recorded. Anthropometric measurements were obtained using standard protocols. Height was measured using a calibrated stadiometer, and weight was recorded using a conventional weighing scale, with children wearing minimal clothing.

For biochemical analysis, a fasting venous blood sample of 3 mL was collected from each participant after an overnight fast. Serum lipid profile parameters measured included total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG), which were analyzed using standard enzymatic methods (cholesterol oxidase–peroxidase method for TC, direct enzymatic method for HDL-C, and glycerol peroxidase method for TG). Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald formula.

Sample size was calculated based on previously published data by Bhadran et al,[14] Considering a 95% confidence interval and 80% study power, the minimum required sample size was 38 participants in each group. Accordingly, the study included a total of 76 participants, with 38 cases and 38 controls.

The study protocol was approved by the Institutional Ethics Committee. Written informed consent was obtained from parents or guardians prior to enrollment. Participant confidentiality was maintained by anonymizing personal and medical information. Parents were informed that participation was voluntary and that they could withdraw at any time without consequences. All procedures were performed with minimal risk, and appropriate medical care was provided if any adverse events occurred.

Data were entered and analyzed using SPSS software and Microsoft Excel. Descriptive statistics were used to present categorical variables as frequencies and percentages, while continuous variables were expressed as mean ± standard deviation or median with range. The independent sample t-test was used to compare mean lipid parameters between cases and controls. Analysis of variance (ANOVA) was applied where necessary. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1: Demographic Characteristics of Study Participants by Drug Group

Variable	Phenytoin (n=38)	Sodium Valproate (n=38)	Levetiracetam (n=38)
Male	20 (52.6%)	22 (57.9%)	14 (36.8%)
Female	18 (47.4%)	16 (42.1%)	24 (63.8%)
Duration <2 yrs	5 (13.2%)	6 (18.4%)	7 (18.4%)
Duration 2–5 yrs	25 (65.8%)	24 (57.9%)	22 (57.9%)
Duration >5 yrs	8 (21.1%)	8 (23.7%)	9 (23.7%)
Positive family history	8 (21.1%)	8 (21.1%)	4 (10.5%)

Table 2: Comparison of Serum Lipid Profile Among Children Receiving Different AED Monotherapies

Parameter (mg/dL)	Phenytoin (Mean ± SD)	Sodium Valproate (Mean ± SD)	Levetiracetam (Mean ± SD)
Total Cholesterol	173.22 ± 36.13	107.74 ± 8.32	125.98 ± 11.31
LDL-C	162.03 ± 29.98	96.53 ± 6.97	89.99 ± 12.37
Triglycerides	131.83 ± 44.30	91.77 ± 8.02	88.67 ± 6.57
HDL-C	48.62 ± 9.94	46.67 ± 5.22	52.25 ± 5.59
VLDL-C	20.62 ± 9.14	21.26 ± 7.30	29.31 ± 7.00

Table 3: Case vs Control Lipid Profile Comparison Within Each Drug Group

Drug	Parameter	Controls (Mean ± SD)	Cases (Mean ± SD)	P value
Phenytoin	Total Cholesterol	113.15 ± 9.74	173.22 ± 36.13	<0.001
	LDL	98.03 ± 7.15	162.03 ± 29.98	<0.001
	Triglycerides	90.62 ± 8.53	131.83 ± 44.30	<0.001
	HDL	60.62 ± 15.13	48.62 ± 9.94	<0.001
	VLDL	21.86 ± 7.75	20.62 ± 9.14	0.268
Levetiracetam	Total Cholesterol	118.88 ± 14.36	125.98 ± 11.31	0.084
	LDL	87.40 ± 12.85	89.99 ± 12.37	0.451
	Triglycerides	90.52 ± 6.67	88.67 ± 6.57	0.289
	HDL	56.50 ± 10.26	52.25 ± 5.59	0.217
	VLDL	30.16 ± 4.71	29.31 ± 7.00	0.741
Sodium Valproate	Total Cholesterol	107.15 ± 9.22	107.74 ± 8.32	0.412
	LDL	95.34 ± 7.91	96.53 ± 6.97	0.841
	Triglycerides	89.47 ± 4.46	91.77 ± 8.02	0.214
	HDL	47.02 ± 4.65	46.67 ± 5.22	0.541
	VLDL	18.63 ± 6.09	21.26 ± 7.30	0.074

- A total of 76 children were included in the study, comprising 38 cases and 38 age- and sex-matched controls in each antiepileptic drug (AED) group.

Phenytoin Group

- In the phenytoin group, the age distribution between cases and controls did not differ significantly ($p = 0.745$). Among controls, the largest proportion of children were aged 9–12 years (31.6%), followed by 6–8 years (23.9%), 3–5 years (23.7%), and <2 years (15.8%). In contrast, the study group showed a higher proportion in the 3–5 years category (52.6%), while 21.1% were <2 years, 15.8% were 9–12 years, and 10.5% were 6–8 years. Gender distribution was comparable between groups, with males constituting 52.6% of cases and 36.8% of controls ($p = 0.356$).
- Regarding duration of epilepsy in the study group, 65.8% had epilepsy for 2–5 years, 21.1% for more than 5 years, and 13.2% for less than 2 years. A positive family history of epilepsy was significantly more common among cases (21.1%) than controls (2.6%) ($p = 0.004$).
- Biochemical analysis revealed significantly higher mean total cholesterol (173.22 ± 36.13 vs 113.15 ± 9.74 mg/dL), LDL (162.03 ± 29.98 vs 98.03 ± 7.15 mg/dL), and triglycerides (131.83 ± 44.30 vs 90.62 ± 8.53 mg/dL) in cases compared to controls (all $p < 0.001$). HDL levels were significantly lower in cases (48.62 ± 9.94 mg/dL) compared to controls (60.62 ± 15.13 mg/dL) ($p < 0.001$). VLDL levels did not show a statistically significant difference ($p = 0.268$).

Levetiracetam Group

- Age distribution between cases and controls was similar ($p = 0.781$). The proportions in <2 years and 3–5 years categories were identical in both groups (15.8% and 23.7%, respectively). Gender distribution was also identical between groups (36.8% males and 63.8% females; $p = 1.0$).
- In the study group, 57.9% had epilepsy for 2–5 years, 23.7% for more than 5 years, and 18.4% for less than 2 years. A positive family history was present in 10.5% of cases and none of the controls ($p = 0.0001$).
- There were no statistically significant differences in lipid parameters between cases and controls. Mean total cholesterol (125.98 ± 11.31 vs 118.88 ± 14.36 mg/dL; $p = 0.084$), LDL (89.99 ± 12.37 vs 87.40 ± 12.85 mg/dL; $p = 0.451$), triglycerides (88.67 ± 6.57 vs 90.52 ± 6.67 mg/dL; $p = 0.289$), HDL (52.25 ± 5.59 vs 56.50 ± 10.26 mg/dL; $p = 0.217$), and VLDL (29.31 ± 7.00 vs 30.16 ± 4.71 mg/dL; $p = 0.741$) were comparable between groups.

Sodium Valproate Group

- Age and gender distributions did not differ significantly between cases and controls (p = 0.514 and p = 0.954, respectively). In the study group, 57.9% had epilepsy for 2–5 years, 23.7% for more than 5 years, and 18.4% for less than 2 years. A positive family history was observed in 21.1% of cases and none of the controls (p = 0.004).
- No statistically significant differences were found between cases and controls in mean total cholesterol (107.74 ± 8.32 vs 107.15 ± 9.22 mg/dL; p = 0.412), LDL (96.53 ± 6.97 vs 95.34 ± 7.91 mg/dL; p = 0.841), triglycerides (91.77 ± 8.02 vs 89.47 ± 4.46 mg/dL; p = 0.214), HDL (46.67 ± 5.22 vs 47.02 ± 4.65 mg/dL; p = 0.541), or VLDL (21.26 ± 7.30 vs 18.63 ± 6.09 mg/dL; p = 0.074).

Comparison Between AED Groups

- Among children receiving AED monotherapy, the highest mean total cholesterol, LDL, and triglyceride levels were observed in the phenytoin group (173.22 ± 36.13 mg/dL, 162.03 ± 29.98 mg/dL, and 131.83 ± 44.30 mg/dL, respectively). HDL levels were highest in the levetiracetam group (52.25 ± 5.59 mg/dL). VLDL levels were highest in the levetiracetam group (29.31 ± 7.00 mg/dL). Gender distribution across AED groups showed a male predominance in the phenytoin and sodium valproate groups, while females predominated in the levetiracetam group.

DISCUSSION

This study aimed to assess the influence of anticonvulsant treatments on lipid profiles in children with seizure disorders. It specifically examined the effects of three widely prescribed antiepileptic drugs—Phenytoin, Levetiracetam, and Sodium Valproate—on serum lipid markers such as total cholesterol (TC), low-density lipoprotein cholesterol (LDLC), high-density lipoprotein cholesterol (HDL-C), triglycerides (TGs), and very low-density lipoprotein cholesterol (VLDL-C). In order to determine the variables that might influence lipid metabolism in children receiving treatment, the study additionally considered clinical features, family history, and demographic information.

The age distribution across different groups in the study highlights variations in the representation of age categories among the control and study groups for three antiepileptic drugs: Phenytoin, Levetiracetam, and Sodium Valproate. This result is consistent with study by Smith et al,[16] who observed that in studies on paediatric epilepsy, age had no discernible impact on lipid metabolism.Similarly, Anju Aggarwal et al,[17] in their study on carbamazepine's effects on serum lipids and liver function, found no major age differences between the cases and controls (mean ages of 8.3±2.8 years and 8.4±2.6 years, respectively). In Pooja Dewan et al.'s,[18] research on phenytoin and valproate, the average age was 7.5±4.4 years across 79 children, with 27 in the valproate group, 25 in the phenytoin group, and 27 in the control group. Similarly, Nadkarni et al,[19] included 95 children (mean age 7.36±2.81 years) and 50 controls (mean age 6.27±2.73 years), showing minimal age variation between the groups.

For the three antiepileptic medications, there were slight variations in the gender distribution between the control and study groups, but none of them were statistically significant. In line with prior research, such as that done by Kumar et al,[20] which found no gender-related variations in lipid outcomes in children with epilepsy, the gender analysis's findings revealed no appreciable alterations in lipid fractions. These consistent findings demonstrate that changes in lipid profiles throughout epilepsy therapy are not significantly influenced by gender.

Most participants had epilepsy for 2–5 years, with similar duration patterns across drug groups. No association was observed between duration of epilepsy and lipid alterations, consistent with Praveen D et al.[21]

A positive family history of epilepsy was significantly more common among cases than controls in all AED groups. While this indicates a hereditary contribution to epilepsy, lipid alterations were more strongly associated with the type of AED rather than family history alone. Bhadran et al,[22] and Katalinic et al,[23] reported that familial cardiovascular risk may amplify AED-induced dyslipidemia, particularly with enzyme-inducing drugs. In contrast, Chaves et al,[24] observed minimal lipid impact with levetiracetam even among genetically predisposed individuals, which supports our findings.

Across the three categories of antiepileptic medications, there were significant variations in the levels of LDL and cholesterol between the study and control groups. Overall, the Phenytoin group exhibited significantly higher cholesterol and LDL levels in the study group, indicating a possible effect of the drug on lipid metabolism. In contrast, the Levetiracetam and Sodium Valproate groups showed minimal or no differences between the study and control groups. Shah et al,[25] conducted a study showing a reduction in cholesterol levels in children treated with enzyme-inducing anticonvulsants like phenytoin and carbamazepine. They suggested that these drugs, by stimulating hepatic enzymes, may alter lipid metabolism, leading to lower cholesterol levels. This result is consistent with our research, which showed that youngsters using anticonvulsants had large drops in their LDL and cholesterol levels. Shah et al,[25] proposed that this reduction in cholesterol could be a compensatory response to increased hepatic clearance, potentially mitigating cardiovascular risks in the long term. LDL levels dramatically dropped when Garcia et al,[26] examined the long-term effects of anticonvulsant treatment on

children's lipid profiles. They found that long-term use of these medications may impact not only cholesterol levels but also other lipoprotein fractions like LDL-C, a critical indicator of the risk of cardiovascular disease. Their study included both more recent medications like valproate and older enzyme-inducing anticonvulsants. This is in line with our findings, which suggest that long-term anticonvulsant therapy may lead to a protective decrease in LDL-C, reducing the incidence of cardiovascular problems and atherosclerosis. However, Nadkarni,[19] discovered that children using anticonvulsants had somewhat higher LDL-C levels, but this difference (p=0.051) was not statistically significant. This may indicate disparities in patient characteristics or individual differences in lipid responses to various anticonvulsant treatments. Although LDL-C was clearly and statistically significantly reduced in our study, Nadkarni's results suggest that anticonvulsants may sometimes have a less noticeable impact on lipid metabolism. Dewan et al,[18] examined the lipid profiles of children treated with various anticonvulsants and found significantly higher LDL-C levels in children on phenytoin compared to those on valproate or the control group. They suggested that phenytoin, as an enzyme-inducing drug, might increase LDL-C levels due to its impact on lipid metabolism. However, our study observed a reduction in both total cholesterol and LDL-C in children treated with phenytoin. This difference could be due to variations in therapy duration, sample size, or lipid profile assessment methods. It's also critical to keep in mind that different people may experience different effects from phenytoin on their lipid levels due to dietary changes, genetic predispositions, and other drugs.

HDL levels for the three antiepileptic drugs varied between the study and control groups, but none of the differences were statistically significant. In conclusion, research participants' HDL levels were somewhat unaltered in the Levetiracetam and Sodium Valproate groups and significantly lower in the Phenytoin group, suggesting that these drugs may have less of an effect on this lipid parameter. Contrasting with our results, Pooja Dewan et al,[18] found higher HDL-C levels in children treated with phenytoin compared to both controls and those treated with valproic acid. This indicates that phenytoin may have an opposite effect on HDL-C, potentially increasing it, while other anticonvulsants generally lower HDL-C. Furthermore, research by Nikkila et al,[27] and O'Neill et al,[28] showed that patients receiving phenytoin had higher HDL-C levels, indicating that it might have a beneficial effect on HDL levels. However, Nadkarni et al,[19] observed significantly lower HDL-C levels in children receiving AEDs compared to controls, with a P-value of 0.000, indicating a clear link between AED therapy and decreased HDL levels. This highlights the potential variation in the impact of anticonvulsants on HDL levels depending on the specific drug and patient factors. Finally, Gopi Srikanth et al,[29] did not report detailed changes in HDL levels but noted that overall lipid profile effects were minimal across different AEDs, which contrasts with the more substantial effects observed in other studies. These discrepancies highlight the complexity of the interactions between anticonvulsant medication and children's lipid metabolism and the need for more study to fully comprehend them.

The Phenytoin group's triglyceride (TGS) levels varied significantly from those of the other medication groups, which showed rather constant levels. These results suggest that phenytoin is strongly associated with elevated triglyceride levels, while levetiracetam and sodium valproate have no influence on triglyceride levels. This raises concerns about phenytoin's effects on lipid metabolism and cardiovascular health. There may be a trend towards greater triglycerides with AEDs, but not necessarily clinically significant in smaller trials, according to the Nadkarni research,[19] which also reported slightly higher triglyceride levels in the case group. Nevertheless, the findings lacked statistical significance (p=0.154). Contrasting with our findings, Gopi Srikanth et al,[29] observed a significant rise in triglyceride levels in children treated with valproate (p=0.001), suggesting that valproate may have a more pronounced effect on triglycerides. They also noticed a near-significant increase in triglyceride levels in children on phenytoin (p=0.053) and a significant rise in levetiracetam-treated children (p=0.013), further highlighting the variability of AEDs in their effects on triglyceride levels.

CONCLUSION

Phenytoin was associated with significant adverse lipid changes in children, while levetiracetam and sodium valproate showed minimal metabolic effects. Regular lipid monitoring should be considered in children receiving enzyme-inducing antiepileptic drugs.

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