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Serum Sodium Changes and Adverse Effect Profile of Carbamazepine Versus Oxcarbazepine in Pediatric Focal Epilepsy: A Randomized Comparative Study

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Abstract

Background: Carbamazepine and oxcarbazepine are commonly used antiseizure medications for pediatric focal epilepsy. Both drugs may alter serum sodium levels and cause adverse effects, but comparative pediatric data, particularly from South Asian settings, remain limited.

Objective: To compare serum sodium changes and adverse-effect profiles of carbamazepine and oxcarbazepine in children with focal epilepsy.

Methods: This prospective, single-center, randomized comparative study was conducted in the Department of Paediatric Neurology, National Institute of Neurosciences and Hospital, Dhaka, from January 2020 to June 2020. Children aged 1 to 15 years with newly diagnosed focal epilepsy were randomized to receive either carbamazepine or oxcarbazepine. Serum sodium levels were measured at baseline, 1 month, 3 months, and 6 months after treatment initiation. Development of hyponatremia, defined as serum sodium ≤135 mEq/L, and adverse effects were assessed during follow-up. Data from 86 children were analyzed, including 42 in the carbamazepine group and 44 in the oxcarbazepine group.

Results: Baseline characteristics were comparable between groups. Mean serum sodium did not differ significantly at baseline, 136.73 ± 1.66 mmol/L with carbamazepine versus 136.09 ± 2.54 mmol/L with oxcarbazepine, $P = .158$, or at 1 month, $P = .845$. At 3 months, serum sodium was significantly lower in the oxcarbazepine group, 137.15 ± 2.49 versus 138.22 ± 2.24 mmol/L, $P = .036$. This difference persisted at 6 months, 137.56 ± 2.54 versus 138.95 ± 2.20 mmol/L, $P = .006$. Hyponatremia at 6 months occurred in 15.9% of oxcarbazepine-treated children and 4.7% of carbamazepine-treated children, $P = .079$. Any adverse reaction occurred in 43.1% and 33.3% of the oxcarbazepine and carbamazepine groups, respectively, $P = .382$. Anorexia was significantly more frequent with oxcarbazepine, 22.7% versus 0.0%, $P = .001$. No treatment-related death or hospitalization occurred.

Conclusion: Oxcarbazepine was associated with lower mean serum sodium than carbamazepine during follow-up, although categorical hyponatremia did not differ significantly. Both drugs were generally tolerated, but serum sodium and adverse-effect monitoring remain important during pediatric focal epilepsy treatment.

Keywords: Carbamazepine, oxcarbazepine, pediatric focal epilepsy, hyponatremia, adverse effects

Introduction

Focal epilepsy is a common and clinically important seizure disorder in childhood, and pharmacological treatment requires a balance between seizure control, drug tolerability, safety monitoring, and long-term adherence. Carbamazepine and oxcarbazepine have both been used widely for focal-onset seizures, with oxcarbazepine developed as a keto-analogue of carbamazepine to provide comparable antiseizure efficacy with potentially improved tolerability. In pediatric focal epilepsy, treatment selection is influenced not only by efficacy but also by drug-specific adverse effects, cost, availability, caregiver adherence, and monitoring feasibility, which are particularly relevant in low- and middle-income clinical settings. A systematic review of approved agents for pediatric focal epilepsy emphasized that medication choice in children should consider both efficacy and tolerability, while a meta-analysis of randomized trials found oxcarbazepine to be an effective treatment option in childhood epilepsy, although adverse-event reporting across studies remained variable.[1,2] More recent pediatric comparative work has also continued to use oxcarbazepine as an active comparator in newly diagnosed focal epilepsy, supporting its ongoing clinical relevance.[3] Among the safety concerns associated with carbamazepine and oxcarbazepine, hyponatremia is one of the most clinically relevant because it may remain asymptomatic, present with nonspecific symptoms, or become clinically significant when severe. The mechanism is usually linked to inappropriate antidiuretic activity and altered renal water handling, but the frequency varies substantially across age groups, study designs, treatment duration, and sodium cut-off values. Pediatric evidence is limited compared with adult data, but Lee et al. reported hyponatremia in 20.8% of epileptic children receiving carbamazepine, oxcarbazepine, or combined therapy, with group-specific rates of 17.9% for carbamazepine and 22.6% for oxcarbazepine.[4] In adult epilepsy cohorts, Yamamoto et al. found severe hyponatremia in 7% of carbamazepine-treated patients, compared with 0.8% in untreated patients and 1.2% in those receiving other antiseizure medications, while a dose above 600 mg/day was associated with markedly higher prevalence.[5] Similarly, Čiauškaitė et al. found hyponatremia in 61.3% of oxcarbazepine-treated patients compared with 5.4% of patients on other antiseizure medications and 3.2% of controls, with longer treatment duration increasing risk. These findings indicate that sodium disturbance is not an incidental laboratory issue but a reproducible drug-related safety concern.[6] Clinical interpretation of carbamazepine- and oxcarbazepine-associated hyponatremia is further complicated by the weak relationship between biochemical abnormality and overt symptoms. Berghuis et al. showed that hyponatremia among people receiving these drugs may be associated with symptoms such as tiredness, instability, dizziness, and cognitive complaints, supporting active clinical inquiry rather than reliance on severe biochemical thresholds alone.[7] Large real-world evidence from Yamamoto et al. also suggests that hyponatremia remains a measurable safety outcome across pediatric, adult, and older epilepsy populations.[8] Beyond sodium disturbance, both carbamazepine and oxcarbazepine may produce gastrointestinal, neurological, dermatological, and systemic adverse reactions. In a tertiary-care adverse drug reaction study, Lee et al. compared carbamazepine- and oxcarbazepine-related adverse reactions and demonstrated overlapping but clinically distinguishable safety profiles.[9] Pediatric pharmacovigilance data also show that adverse reactions to antiseizure medications are common enough to justify systematic monitoring in children with epilepsy.[10] Despite this evidence, randomized pediatric data directly comparing serial serum sodium changes and adverse-effect profiles of carbamazepine and oxcarbazepine remain limited, particularly from South Asian settings where both drugs continue to be used in routine pediatric neurology practice. Existing studies often focus either on efficacy, adult hyponatremia, pharmacovigilance reports, or oxcarbazepine alone, leaving fewer prospective comparative data in children with focal epilepsy.

Therefore, the present randomized comparative study was conducted to evaluate serum sodium changes and adverse effects among children with focal epilepsy receiving carbamazepine or oxcarbazepine over six months of follow-up.

Methods

This open-label, prospective, single-center, randomized comparative study was conducted in the Department of Paediatric Neurology, National Institute of Neurosciences and Hospital, Dhaka, from January 2020 to June 2020. Children aged 1 to 15 years with newly diagnosed focal epilepsy were enrolled after clinical assessment, seizure semiology review, and electroencephalographic confirmation by focal or multifocal epileptic discharges or compatible focal slowing. Children were excluded before enrollment if they had prior antiseizure medication exposure, mixed seizure types, suspected neurometabolic or neurodegenerative disease, structural brain lesion on cranial CT or MRI, congenital or acquired heart disease, renal or hepatic impairment, or abnormal baseline serum sodium. After written informed consent from parents or caregivers, eligible participants were randomized in a 1:1 ratio by lottery method to receive either carbamazepine or oxcarbazepine. Carbamazepine was started at 5 mg/kg/day orally at night for 7 days, then increased to 10 mg/kg/day in two divided doses, with further titration by 5 mg/kg/week according to seizure control, up to a maximum of 25 mg/kg/day. Oxcarbazepine was started at 5 mg/kg/day orally at night for 7 days, then increased to 10 mg/kg/day in two divided doses, with further titration by 5 mg/kg/week according to seizure control, up to a maximum of 40 mg/kg/day. Baseline evaluation included demographic and clinical variables, seizure frequency, EEG, cranial neuroimaging, serum electrolytes, and relevant biochemical tests. Participants were followed clinically and biochemically at 1 month, 3 months, and 6 months after treatment initiation. Children who developed major adverse reactions, inadequate clinical response, or increased seizure frequency after treatment initiation were withdrawn from per-protocol analysis and described in the participant flow diagram [Figure 1]. The primary outcome was serum sodium change during follow-up, and secondary outcomes included development of hyponatremia, defined as serum sodium ≤135 mEq/L, seizure frequency, and adverse effects. Data were analyzed using SPSS version 22.0. Continuous variables were expressed as mean ± standard deviation and compared using independent-samples t-test. Categorical variables were expressed as frequency and percentage and compared using chi-square test or Fisher's exact test, as appropriate. Risk differences, risk ratios, and 95% confidence intervals were calculated for categorical outcomes where applicable. A *P*-value <0.05 was considered statistically significant. Ethical clearance was obtained from the Ethical Review Committee of the National Institute of Neurosciences and Hospital, and confidentiality and voluntary participation were maintained throughout the study. Trial registration status was not available in the source thesis documentation and should be reported according to the target journal's requirement.

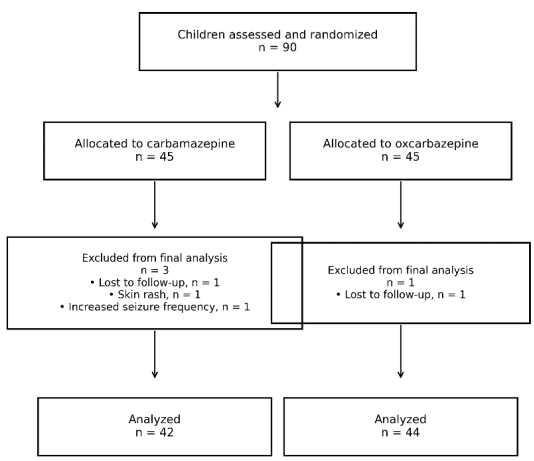

Figure 1: Participant flow diagram.

Results

A total of 90 children with newly diagnosed focal epilepsy were randomized equally to receive carbamazepine or oxcarbazepine. Four children were excluded from the final analysis, three from the carbamazepine group and one from the oxcarbazepine group. The final analyzed population included 86 children, with 42 in the carbamazepine group and 44 in the oxcarbazepine group. Baseline demographic and clinical characteristics were comparable between groups [Table 1].

The two treatment groups were comparable at baseline. Mean age was 8.08 ± 2.45 years in the carbamazepine group and 7.50 ± 2.51 years in the oxcarbazepine group, P = .275. Baseline seizure frequency was also similar, 2.51 ± 1.18 versus 2.91 ± 1.38 episodes per month, P = .141. Most children in both groups had focal seizures without awareness, 92.8% in the carbamazepine group and 81.8% in the oxcarbazepine group [Table 2].

Serum sodium was comparable at baseline and 1 month between the two groups. At 3 months, the oxcarbazepine group had significantly lower mean serum sodium than the carbamazepine group, 137.15 ± 2.49 versus 138.22 ± 2.24 mmol/L, mean difference -1.07 mmol/L, P = .036. At 6 months, this difference became more pronounced, 137.56 ± 2.54 versus 138.95 ± 2.20 mmol/L, mean difference -1.39 mmol/L, P = .006. This indicates that the sodium difference emerged during continued therapy rather than immediately after treatment initiation [Table 3].

Hyponatremia occurred in both groups during follow-up. At 1 month, the frequency was similar, 19.0% in the carbamazepine group and 15.9% in the oxcarbazepine group, P = .777. At 3 months, hyponatremia was numerically more frequent with oxcarbazepine, 25.0% versus 16.6%, P = .292. At 6 months, 15.9% of oxcarbazepine-treated children and 4.7% of carbamazepine-treated children developed hyponatremia, giving an approximate risk ratio of 3.34, although the difference did not reach statistical significance, P = .079 [Table 4].

Any adverse reaction was reported in 33.3% of children receiving carbamazepine and 43.1% receiving oxcarbazepine, with no statistically

Table 1: Baseline demographic and clinical characteristics of the study participants

Characteristic	Carbamazepine, *n* = 42	Oxcarbazepine, *n* = 44	*P*
Age group, years, *n* (%)			
1 to 5	7 (16.6)	10 (22.7)	
6 to 10	29 (69.0)	29 (65.9)	
11 to 15	6 (14.2)	5 (11.3)	
Age, years, mean ± SD	8.08 ± 2.45	7.50 ± 2.51	.275
Sex, *n* (%)			
Male	22 (52.3)	26 (59.1)	.396
Female	20 (47.6)	18 (40.9)	
Age at seizure onset, years, mean ± SD	5.36 ± 2.10	6.20 ± 2.45	.083
Baseline seizure frequency/month, mean ± SD	2.51 ± 1.18	2.91 ± 1.38	.141
Focal seizure pattern, *n* (%)			
With awareness	3 (7.1)	8 (18.1)	.108
Without awareness	39 (92.8)	36 (81.8)	

Note. Values are presented as mean ± SD or *n* (%). SD: standard deviation. *P*-values were derived using unpaired t-test for continuous variables and chi-square test for categorical variables.

Table 2: Serum sodium concentration at baseline and follow-up

Time point	Carbamazepine, *n* = 42, mean ± SD	Oxcarbazepine, *n* = 44, mean ± SD	Mean difference, OXC minus CBZ	95% CI for mean difference	*P*
Baseline	136.73 ± 1.66	136.09 ± 2.54	–0.64	–1.56 to 0.28	.158
1 month	138.07 ± 2.61	138.18 ± 2.77	0.11	–1.04 to 1.26	.845
3 months	138.22 ± 2.24	137.15 ± 2.49	–1.07	–2.08 to –0.06	.036
6 months	138.95 ± 2.20	137.56 ± 2.54	–1.39	–2.41 to –0.37	.006

Note. Serum sodium is expressed as mmol/L. CI: confidence interval; OXC: oxcarbazepine; CBZ: carbamazepine. *P*-values are from unpaired t-tests.

Table 3: Development of hyponatremia during follow-up

Follow-up time	Carbamazepine, *n* = 42, *n* (%)	Oxcarbazepine, *n* = 44, *n* (%)	Risk difference, OXC - CBZ	Risk ratio	95% CI for risk ratio	*P*
1 month	8 (19.0)	7 (15.9)	–3.1%	0.84	0.33 to 2.10	.777
3 months	7 (16.6)	11 (25.0)	8.4%	1.50	0.64 to 3.50	.292
6 months	2 (4.7)	7 (15.9)	11.2%	3.34	0.74 to 15.18	.079

Note. Hyponatremia was defined as serum sodium ≤135 mEq/L. CI: confidence interval; OXC: oxcarbazepine; CBZ: carbamazepine.

Table 4: Adverse effect profile after 6 months of therapy

Adverse effect	Carbamazepine, *n* = 42, *n* (%)	Oxcarbazepine, *n* = 44, *n* (%)	Risk difference, OXC minus CBZ	Risk ratio	95% CI for risk ratio	*P*
Any adverse reaction	14 (33.3)	19 (43.1)	9.8%	1.30	0.75 to 2.24	.382
Anorexia	0 (0.0)	10 (22.7)	22.7%	Not estimable	Not estimable	.001
Vomiting	6 (14.2)	1 (2.2)	–12.0%	0.16	0.02 to 1.27	.110
Nausea	4 (9.5)	3 (6.8)	–2.7%	0.72	0.17 to 3.01	1.000
Headache	2 (4.7)	1 (2.2)	–2.5%	0.48	0.04 to 5.07	1.000
Irritability	1 (2.3)	0 (0.0)	–2.3%	Not estimable	Not estimable	1.000
Drowsiness/ sleepiness	0 (0.0)	2 (4.5)	4.5%	Not estimable	Not estimable	.494
Malaise	1 (2.3)	1 (2.2)	–0.1%	0.95	0.06 to 14.77	1.000
Vertigo	0 (0.0)	1 (2.2)	2.2%	Not estimable	Not estimable	1.000

Note. CI: confidence interval; OXC: oxcarbazepine; CBZ: carbamazepine.

significant overall difference, $P = .382$. Anorexia was the only clearly significant specific adverse effect, occurring in 22.7% of oxcarbazepine-treated children and none of the carbamazepine-treated children, $P = .001$. Vomiting was numerically more frequent in the carbamazepine group, 14.2% versus 2.2%, but this difference was not statistically significant, $P = .110$. Other adverse effects were infrequent, and no death or hospitalization attributable to trial treatment was reported.

Discussion

The present randomized comparative study showed that oxcarbazepine was associated with relatively lower serum sodium than carbamazepine

during continued follow-up in children with focal epilepsy. Baseline sodium levels were comparable between groups, 136.73 ± 1.66 mmol/L in the carbamazepine group and 136.09 ± 2.54 mmol/L in the oxcarbazepine group, $P = 0.158$. No difference was observed at 1 month, but by 3 months, mean serum sodium was significantly lower with oxcarbazepine, 137.15 ± 2.49 versus 138.22 ± 2.24 mmol/L, $P = 0.036$. This difference persisted at 6 months, 137.56 ± 2.54 versus 138.95 ± 2.20 mmol/L, $P = 0.006$. These findings suggest that the sodium-lowering tendency of oxcarbazepine may become more apparent after sustained exposure rather than immediately after treatment initiation. This observation is consistent with previous pediatric and adult epilepsy literature. Lee et al. reported hyponatremia in 20.8% of children receiving carbamazepine, oxcarbazepine, or combined therapy, with rates of 17.9% among carbamazepine users and 22.6% among oxcarbazepine users.[4] Although the difference between drugs was modest in that pediatric study, it supports the present finding that sodium disturbance occurs with both agents but may be more frequent with oxcarbazepine. In a larger epilepsy cohort, Berghuis et al. found hyponatremia in 26% of carbamazepine users and 46% of oxcarbazepine users, with severe hyponatremia in 7% and 22%, respectively.[11] Čiauškaitė et al. also reported a high frequency of hyponatremia among oxcarbazepine-treated patients, 61.3%, and found that longer treatment duration was associated with lower serum sodium.[6] The lower absolute frequency in the present study is likely explained by the pediatric age group, monotherapy design, exclusion of children with abnormal baseline sodium or comorbidity, and the shorter 6-month follow-up. The categorical hyponatremia outcome in this study followed the same direction but did not reach statistical significance. At 6 months, hyponatremia occurred in 15.9% of children receiving oxcarbazepine compared with 4.7% receiving carbamazepine, $P = 0.079$, with an approximate risk ratio of 3.34. This result is clinically relevant despite statistical non-significance because the event count was small and the confidence interval was wide. Yamamoto et al., using large real-world epilepsy data, showed that moderate-severe hyponatremia is less frequent in pediatric patients than in adults and older adults, which may partly explain the absence of a strong categorical signal in this cohort.[8] Berghuis et al. further emphasized that carbamazepine- and oxcarbazepine-associated hyponatremia may be symptomatic even when clinical manifestations are nonspecific, supporting routine biochemical monitoring alongside clinical questioning.[7] The adverse-effect profile in this study showed broadly comparable overall tolerability. Any adverse reaction was reported in 33.3% of the carbamazepine group and 43.1% of the oxcarbazepine group, $P = 0.382$. This aligns with Lee et al., who found no significant overall difference in adverse drug reaction incidence between carbamazepine and oxcarbazepine, although the pattern of reactions differed between the two drugs.[9] Pediatric safety studies have also shown that adverse reactions to antiseizure medications are common but often mild. Anderson et al. reported adverse drug reactions in 31% of children receiving antiepileptic drugs, while Egunsola et al. demonstrated the value of active adverse-effect elicitation in pediatric epilepsy practice.[12,13] The most distinct adverse-effect finding in the present study was anorexia, occurring in 22.7% of oxcarbazepine-treated children and none of the carbamazepine-treated children, $P = 0.001$. This finding should be interpreted cautiously because anorexia is not consistently reported as the dominant oxcarbazepine-related adverse effect in larger pharmacovigilance studies, where dermatological, neurological, and gastrointestinal events are more commonly emphasized.[9,10] However, in a pediatric Bangladeshi clinical setting, appetite-related symptoms may be particularly important for adherence, caregiver concern, and nutritional monitoring. Vomiting was numerically more frequent with carbamazepine, 14.2% versus 2.2%, but this difference was not statistically significant. Other adverse effects were infrequent, and no treatment-related death or hospitalization occurred, supporting the short-term tolerability of both drugs. Overall, the study indicates that both carbamazepine and oxcarbazepine were generally

tolerated in children with focal epilepsy over 6 months, but oxcarbazepine was associated with significantly lower mean serum sodium and a numerically higher rate of hyponatremia. These findings support periodic sodium monitoring, particularly during continued oxcarbazepine therapy, while also reinforcing the need for structured adverse-effect assessment in pediatric epilepsy follow-up.

Limitations of the Study

This study was conducted in a single tertiary-care center with a relatively small sample size and a short follow-up period of six months, which may limit generalizability and reduce power to detect less frequent adverse effects or statistically significant differences in hyponatremia. The study was not blinded, and adverse effects were assessed clinically without standardized severity grading or formal causality scoring.

Conclusion

Oxcarbazepine was associated with significantly lower mean serum sodium levels than carbamazepine at 3 and 6 months of therapy among children with focal epilepsy. Although hyponatremia was numerically more frequent in the oxcarbazepine group, the difference was not statistically significant. Both drugs were generally well tolerated over six months, with no treatment-related death or hospitalization; however, anorexia was significantly more common among children receiving oxcarbazepine. These findings support periodic serum sodium monitoring, particularly during oxcarbazepine therapy, along with structured clinical assessment of adverse effects during pediatric epilepsy follow-up.

Ethical Approval

The study was approved by the Institutional Ethics Committee.

References

1. Arya R, Glauser TA. Pharmacotherapy of focal epilepsy in children: a systematic review of approved agents. CNS Drugs 2013 Apr;27(4):273–86. doi:10.1007/s40263-013-0048-z PubMed PMID: 23515971; PubMed Central PMCID: PMC5768201.
2. Geng H, Wang C. Efficacy and safety of oxcarbazepine in the treatment of children with epilepsy: a meta-analysis of randomized controlled trials. Neuropsychiatr Dis Treat 2017 Mar 2;13:685–95. doi:10.2147/NDT.S130269 PubMed PMID: 28293110; PubMed Central PMCID: PMC5342615.
3. Yi JQ, Huang S, Wu MJ, Ma JH, Huang LJ, Liang S, et al. Comparison of the effectiveness and safety of perampanel and oxcarbazepine as monotherapy in children and adolescents with newly diagnosed focal epilepsy. Front Pharmacol 2023 Aug 30;14. doi:10.3389/fphar.2023.1189058
4. Lee KH, Song JH, Cha SH, Chung SJ. The age and dose-related hyponatremia during carbamazepine and oxcarbazepine therapy in epileptic children. Korean J Pediatr 2008;409–14.
5. Yamamoto Y, Takahashi Y, Imai K, Ohta A, Kagawa Y, Inoue Y. Prevalence and risk factors for hyponatremia in adult epilepsy patients: large-scale cross-sectional cohort study. Seizure 2019 Dec;73:26–30. doi:10.1016/j.seizure.2019.10.013 PubMed PMID: 31707295.
6. Čiauškaitė J, Gelžinienė G, Jurkevičienė G. Oxcarbazepine and hyponatremia. Medicina 2022 Apr 19;58(5):559. doi:10.3390/medicina58050559
7. Berghuis B, Hulst J, Sonsma A, McCormack M, de Haan GJ, Sander JW, et al. Symptomatology of carbamazepine- and oxcarbazepine-induced hyponatremia in people with epilepsy. Epilepsia 2021;62(3):778–84. doi:10.1111/epi.16828
8. Yamamoto Y, Ohta A, Usui N, Imai K, Kagawa Y, Takahashi Y. Incidence trends and risk factors for hyponatremia in epilepsy patients: A large-scale real-world data study. Heliyon 2023 Jul 26;9(8):e18721. doi:10.1016/j.heliyon.2023.e18721 PubMed PMID: 37554799; PubMed Central PMCID: PMC10404753.
9. Lee JE, Min KR, Kim SH, Kim AH, Kim ST. Analysis of adverse drug reactions with carbamazepine and oxcarbazepine at a tertiary care hospital. Yonsei Med J 2020 Oct 1;61(10):875–9. doi:10.3349/ymj.2020.61.10.875 PubMed PMID: 32975062; PubMed Central PMCID: PMC7515781.
10. Hernández García E, Naranjo L, Pichardo-Macías LA, Bernad Bernad MJ, Castro-Pastrana LI, Ruiz García M, et al. Analysis of adverse drug reactions in pediatric

patients with epilepsy: an intensive pharmacovigilance study. Children 2023;10(11):1775.

11. Berghuis B, van der Palen J, de Haan GJ, Lindhout D, Koeleman BPC, Sander JW, et al. Carbamazepine- and oxcarbazepine-induced hyponatremia in people with epilepsy. Epilepsia 2017;58(7):1227–33. doi:10.1111/epi.13777
12. Anderson M, Egunsola O, Cherrill J, Millward C, Fakis A, Choonara I. A prospective study of adverse drug reactions to antiepileptic drugs in children. BMJ Open 2015 May 1;5(6):e008298. doi:10.1136/bmjopen-2015-008298
13. Egunsola O, Choonara I, Sammons HM, Whitehouse WP. Safety of antiepileptic drugs in children and young people: a prospective cohort study. Seizure 2018 Mar;56:20–5. doi:10.1016/j.seizure.2018.01.018 PubMed PMID: 29427834.

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