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Original Research Article

Serum sodium changes in children with focal epilepsy receiving carbamazepine or oxcarbazepine therapy: a randomized controlled trial

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ABSTRACT

Background: Carbamazepine and oxcarbazepine are commonly used antiseizure medications for focal epilepsy in children. Both drugs may alter serum sodium, but comparative pediatric data, particularly from South Asian settings, remain limited. The objective of this study was to compare serum sodium changes, the incidence of hyponatremia, and adverse effect profiles between children with focal epilepsy receiving carbamazepine and those receiving oxcarbazepine therapy.
Methods: This prospective, single-centre, open-label randomized controlled trial was conducted in the Department of Paediatric Neurology, National Institute of Neurosciences and Hospital, Dhaka, Bangladesh, from January to June 2020. Children aged 1 to 15 years with newly diagnosed focal epilepsy were randomized to receive either carbamazepine or oxcarbazepine. Serum sodium was measured before treatment and at 1 month, 3 months, and 6 months after treatment initiation. Hyponatremia was defined as serum sodium ≤135 mEq/l, and severe hyponatremia as serum sodium ≤128 mEq/l. The final analysis included 86 children, 42 in the carbamazepine group and 44 in the oxcarbazepine group.
Results: Baseline serum sodium was similar between groups, 136.73±1.66 mmol/l with carbamazepine and 136.09±2.54 mmol/l with oxcarbazepine, p=0.158. At 1 month, serum sodium remained comparable, p=0.845. Mean serum sodium was significantly lower in the oxcarbazepine group at 3 months, 137.15±2.49 versus 138.22±2.24 mmol/l, p=0.036, and at 6 months, 137.56±2.54 versus 138.95±2.20 mmol/l, p=0.006. Hyponatremia at 6 months occurred in 15.9% of children receiving oxcarbazepine and 4.8% receiving carbamazepine, risk ratio 3.34, 95% CI 0.74 to 15.18, p=0.157. Severe hyponatremia occurred in one child in the oxcarbazepine group, and no symptomatic hyponatremia, hospitalization, or treatment-attributable death was reported.
Conclusions: Oxcarbazepine was associated with significantly lower mean serum sodium than carbamazepine at 3 and 6 months, although categorical hyponatremia was not significantly different. Routine serum sodium monitoring should be considered during oxcarbazepine therapy in children with focal epilepsy.

Keywords: Focal epilepsy, Children, Carbamazepine, Oxcarbazepine, Hyponatremia

INTRODUCTION

Epilepsy is a major neurological disorder in childhood and remains an important public health concern in Bangladesh. In a national household survey, Mohammad et al reported an epilepsy prevalence of 8.4 per 1000 population in Bangladesh, with a similar burden among children, supporting the need for locally generated

pediatric epilepsy evidence.[1] Focal-onset seizures constitute a clinically important subgroup of childhood epilepsy because treatment selection depends on seizure type, epilepsy syndrome, age, comorbidities, tolerability, and long-term safety considerations. Contemporary reviews of pediatric focal-onset seizure management emphasize that antiseizure medication choice should be individualized, with increasing attention to etiology, developmental profile, and adverse-effect burden rather than seizure suppression alone.[2] A systematic review of approved pharmacological agents for focal epilepsy in children identified oxcarbazepine as an evidence-supported option for pediatric partial-onset seizures, while carbamazepine has remained widely used as a conventional sodium-channel antiseizure medication in focal epilepsy.[3] Evidence syntheses have also supported the clinical utility of oxcarbazepine in children with epilepsy. Geng and Wang, in a meta-analysis of randomized controlled trials, reported that oxcarbazepine was effective and generally safe in pediatric epilepsy, while Liu et al found comparable efficacy and overall adverse reaction rates between levetiracetam and oxcarbazepine monotherapy in children, although the included studies varied in epilepsy type, duration, and adverse-event reporting.[4,5] Despite these therapeutic advantages, sodium-channel antiseizure medications, particularly carbamazepine and oxcarbazepine, are clinically relevant causes of hyponatremia. Oxcarbazepine has been repeatedly associated with reduced serum sodium, and Čiauškaitė et al highlighted that oxcarbazepine-related hyponatremia may be frequent, sometimes asymptomatic, and more likely with longer treatment duration.[6] In a large cohort of people with epilepsy receiving carbamazepine or oxcarbazepine, Berghuis et al reported that hyponatremia was common in both treatment groups and more frequent with oxcarbazepine than carbamazepine.[7] Another subsequent study by Berghuis et al from 2021 further showed that carbamazepine- and oxcarbazepine-induced hyponatremia was associated with clinically relevant symptoms, including dizziness, tiredness, instability, and diplopia, indicating that even medication-related sodium reductions may have functional consequences.[8] Large real-world data also support the broader clinical relevance of this problem. Yamamoto et al identified hyponatremia as an important adverse biochemical outcome among patients with epilepsy and emphasized the role of antiseizure medication exposure and patient-related factors in hyponatremia risk.[9] Pediatric-specific evidence is more limited, but recent trial data have strengthened concern regarding oxcarbazepine-associated sodium changes in children. In the SCHO randomized controlled trial, Neha et al evaluated oral sodium chloride supplementation among children receiving oxcarbazepine monotherapy and found that supplementation reduced the incidence of hyponatremia, although symptomatic and severe hyponatremia were not significantly reduced.[10] These findings indicate that sodium disturbance during oxcarbazepine therapy is sufficiently important to justify preventive and monitoring strategies in children. However, direct pediatric comparative data on serum sodium changes during carbamazepine versus oxcarbazepine therapy remain limited, especially in South Asian clinical settings where dietary sodium intake, nutritional status, follow-up practices, and access to biochemical monitoring may differ from high-income settings. Therefore, this randomized controlled trial was conducted to compare serum sodium levels, development of hyponatremia, seizure-frequency outcomes, and adverse effects among Bangladeshi children with focal epilepsy receiving carbamazepine or oxcarbazepine therapy over 6 months.

METHODS

This prospective, single-centre, open-label randomized controlled trial was conducted in the Department of Paediatric Neurology, National Institute of Neurosciences and Hospital, Dhaka, Bangladesh, from January to June 2020. Children aged 1 to 15 years with newly diagnosed focal epilepsy, with or without awareness, were enrolled after clinical assessment, seizure semiology review, and electroencephalographic evaluation. Focal epilepsy was diagnosed based on suggestive seizure semiology, video documentation when available, and compatible EEG findings such as focal or multifocal epileptiform discharges or focal polymorphic delta slowing. Children were excluded if they had previous antiepileptic drug exposure, mixed seizure types, suspected neurometabolic or neurodegenerative disease, structural brain lesions on CT or MRI, congenital or acquired heart disease, renal or hepatic impairment, abnormal baseline serum sodium, inadequate clinical response, or increased seizure frequency after treatment initiation. Eligible participants were randomly assigned in a 1:1 ratio to receive either carbamazepine or oxcarbazepine using a lottery method with sealed envelopes. 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 until seizure control was achieved, up to a maximum dose 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 until seizure control was achieved, up to a maximum dose of 40 mg/kg/day. Baseline evaluation included clinical history, physical and neurological examination, EEG, neuroimaging when indicated, serum electrolytes, and relevant laboratory tests including serum creatinine and alanine aminotransferase. Participants were followed clinically and biochemically at 1 month, 3 months, and 6 months after treatment initiation. Seizure frequency was recorded using caregiver-maintained seizure diaries, and adverse events were assessed at each follow-up. Children who developed major adverse reactions, such as skin rash or jaundice, or increased seizure frequency were withdrawn from the trial. The primary outcome was change in serum sodium level during 6 months of therapy. Secondary outcomes included development of hyponatremia, severe hyponatremia, seizure frequency

during follow-up, and treatment-emergent adverse effects. Hyponatremia was defined as serum sodium ≤135 mEq/l, and severe hyponatremia as serum sodium ≤128 mEq/l. Ethical approval was obtained from the Ethical Review Committee of the National Institute of Neurosciences and Hospital. Written informed consent was obtained from parents or legal guardians before enrolment, and confidentiality was maintained throughout the study. Data were analyzed using SPSS version 22.0. Continuous variables were summarized as mean±standard deviation and compared using the unpaired t test. Categorical variables were summarized as frequency and percentage and compared using the chi-square test or Fisher's exact test when cell counts were small. Risk differences and risk ratios were calculated for hyponatremia outcomes. A two-sided p value <0.05 was considered statistically significant.

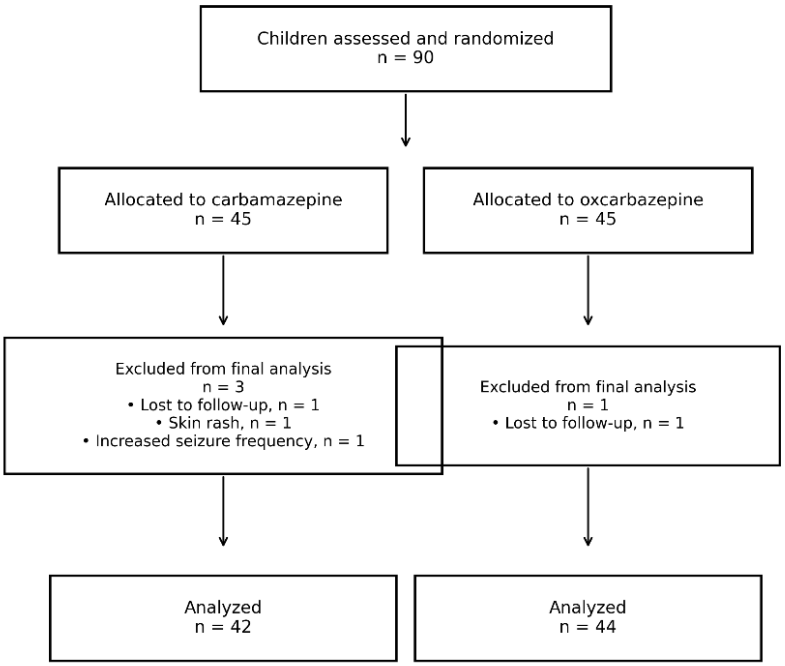

Figure 1: Patient flow diagram.

RESULTS

Baseline demographic and clinical characteristics were generally balanced between the two treatment groups. The mean age was 8.08±2.45 years in the carbamazepine group and 7.50±2.51 years in the oxcarbazepine group. Most children were aged 6–10 years in both groups, 69.0% and 65.9%, respectively. Male participants accounted for 52.3% in the carbamazepine group and 59.1% in the oxcarbazepine group. Mean age at seizure onset was 5.36±2.10 years versus 6.20±2.45 years, and pretreatment seizure frequency was 2.51±1.18 versus 2.91±1.38 seizures per month. Focal seizures without awareness were the predominant seizure pattern in both groups, 92.8% in the carbamazepine group and 81.8% in the oxcarbazepine group (Table 1).

Baseline serum sodium was similar 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. At 1 month, serum sodium remained comparable, 138.07±2.61 versus 138.18±2.77 mmol/l, p=0.845. However, mean serum sodium was significantly lower in the oxcarbazepine group at 3 months, 137.15±2.49 versus 138.22±2.24 mmol/l, mean difference 1.07 mmol/l, 95% CI 0.05 to 2.09, p=0.036, and at 6 months, 137.56±2.54 versus 138.95±2.20 mmol/l, mean difference 1.39 mmol/l, 95% CI 0.37 to 2.41, p=0.006 (Table 2).

The proportion of children developing hyponatremia did not differ significantly between treatment groups at any follow-up point. At 1 month, hyponatremia occurred in 19.0% of children receiving carbamazepine and 15.9% receiving oxcarbazepine, risk ratio 0.84, 95% CI 0.33 to 2.10, p=0.781. At 3 months, the proportion was higher in the oxcarbazepine group, 25.0% versus 16.7%, risk ratio 1.50, 95% CI 0.64 to 3.50, p=0.430. At 6 months, hyponatremia was observed in 15.9% of the oxcarbazepine group compared with 4.8% of the carbamazepine group, risk ratio 3.34, 95% CI 0.74 to 15.18, p=0.157 (Table 3).

Any adverse effect was reported in 33.3% of children in the carbamazepine group and 43.2% in the oxcarbazepine group, p=0.382. Anorexia was significantly more frequent with oxcarbazepine, 22.7% versus 0.0%, p=0.001. Vomiting was numerically more common with carbamazepine, 14.3% versus 2.3%, but this difference was not statistically significant, p=0.110. Other adverse

effects were infrequent and did not differ significantly between groups. Severe hyponatremia occurred in one child in the oxcarbazepine group and none in the carbamazepine group. No symptomatic hyponatremia, treatment-attributable hospitalization, or treatment-attributable death was reported. One child in the carbamazepine group withdrew due to skin rash, and one withdrew due to increased seizure frequency (Table 4).

Table 1: Baseline demographic and clinical characteristics of children with focal epilepsy by treatment group.

Variables	Carbamazepine, (n=42)	Oxcarbazepine, (n=44)
	N (%)	N (%)
Age group (years)		
1-5	7 (16.6)	10 (22.7)
6-10	29 (69.0)	29 (65.9)
11-15	6 (14.2)	5 (11.3)
Mean±SD	8.08±2.45	7.50±2.51
Sex		
Male	22 (52.3)	26 (59.1)
Female	20 (47.6)	18 (40.9)
Age at onset of seizure (years)		
Mean±SD	5.36±2.10	6.20±2.45
Pretreatment seizure frequency per month		
Mean±SD	2.51±1.18	2.91±1.38
Pattern of focal seizure		
With awareness	3 (7.1)	8 (18.1)
Without awareness	39 (92.8)	36 (81.8)

Table 2: Serum sodium levels during 6-month follow-up by treatment group.

Variables	Carbamazepine, n=42, mean±SD	Oxcarbazepine, n=44, mean±SD	Mean difference	95% CI	P value
Serum sodium level, mmol/l					
Before treatment	136.73±1.66	136.09±2.54	0.64	-0.28 to 1.56	0.158
After 1 month	138.07±2.61	138.18±2.77	-0.11	-1.26 to 1.04	0.845
After 3 months	138.22±2.24	137.15±2.49	1.07	0.05 to 2.09	0.036
After 6 months	138.95±2.20	137.56±2.54	1.39	0.37 to 2.41	0.006

Note. Serum sodium is expressed in mmol/l; for sodium, mmol/l and mEq/l are numerically equivalent. Mean difference was calculated as carbamazepine minus oxcarbazepine.

Table 3: Development of hyponatremia during follow-up by treatment group.

Follow-up time points	Carbamazepine, (n=42) N (%)	Oxcarbazepine, (n=44) N (%)	Risk difference, OXC minus CBZ, percentage points, 95% CI	Risk ratio, OXC vs CBZ, 95% CI	Fisher's exact p value
After 1 month	8 (19.0)	7 (15.9)	-3.1 (-19.2 to 12.9)	0.84 (0.33 to 2.10)	0.781
After 3 months	7 (16.7)	11 (25.0)	8.3 (-8.7 to 25.4)	1.50 (0.64 to 3.50)	0.430
After 6 months	2 (4.8)	7 (15.9)	11.1 (-1.4 to 23.7)	3.34 (0.74 to 15.18)	0.157

Note. Risk difference was calculated as oxcarbazepine percentage minus carbamazepine percentage.

Table 4: Adverse effects reported during 6-month follow-up by treatment group.

Safety outcomes	Analysis population	Carbamazepine	Oxcarbazepine	Risk difference (OXC–CBZ)	P value
Any reported adverse effect	Analyzed sample	14/42 (33.3)	19/44 (43.2)	9.8	0.382
Gastrointestinal adverse effects					
Anorexia	Analyzed sample	0/42 (0.0)	10/44 (22.7)	22.7	0.001
Vomiting	Analyzed sample	6/42 (14.3)	1/44 (2.3)	-12.0	0.110
Nausea	Analyzed sample	4/42 (9.5)	3/44 (6.8)	-2.7	1.000

Continued.

Safety outcomes	Analysis population	Carbamazepine	Oxcarbazepine	Risk difference (OXC–CBZ)	P value
Neurological or neurobehavioral adverse effects					
Headache	Analyzed sample	2/42 (4.8)	1/44 (2.3)	-2.5	1.000
Irritability	Analyzed sample	1/42 (2.4)	0/44 (0.0)	-2.4	1.000
Drowsiness or sleepiness	Analyzed sample	0/42 (0.0)	2/44 (4.5)	4.5	0.494
Vertigo	Analyzed sample	0/42 (0.0)	1/44 (2.3)	2.3	1.000
Malaise	Analyzed sample	1/42 (2.4)	1/44 (2.3)	-0.1	1.000
Sodium-related safety outcomes					
Severe hyponatremia	Analyzed sample	0/42 (0.0)	1/44 (2.3)	2.3	1.000
Symptomatic hyponatremia	Analyzed sample	0/42 (0.0)	0/44 (0.0)	0.0	Not estimable
Hospitalization attributable to trial treatment	Randomized/exposed sample	0/45 (0.0)	0/45 (0.0)	0.0	Not estimable
Treatment-attributable death	Randomized/exposed sample	0/45 (0.0)	0/45 (0.0)	0.0	Not estimable
Treatment discontinuation or withdrawal					
Skin rash leading to withdrawal	Randomized/exposed sample	1/45 (2.2)	0/45 (0.0)	-2.2	1.000
Increased seizure frequency leading to withdrawal	Randomized/exposed sample	1/45 (2.2)	0/45 (0.0)	-2.2	1.000

Note. P values for individual adverse effects were reported using Fisher's exact test.

Table 5: Descriptive change in mean serum sodium from baseline.

Variables	Carbamazepine mean sodium, mmol/l	Change from baseline, mmol/l	Oxcarbazepine mean sodium, mmol/l	Change from baseline, mmol/l	Difference in mean change, CBZ minus OXC, mmol/l
Baseline	136.73	---	136.09	---	---
1 month	138.07	+1.34	138.18	+2.09	-0.75
3 months	138.22	+1.49	137.15	+1.06	+0.43
6 months	138.95	+2.22	137.56	+1.47	+0.75

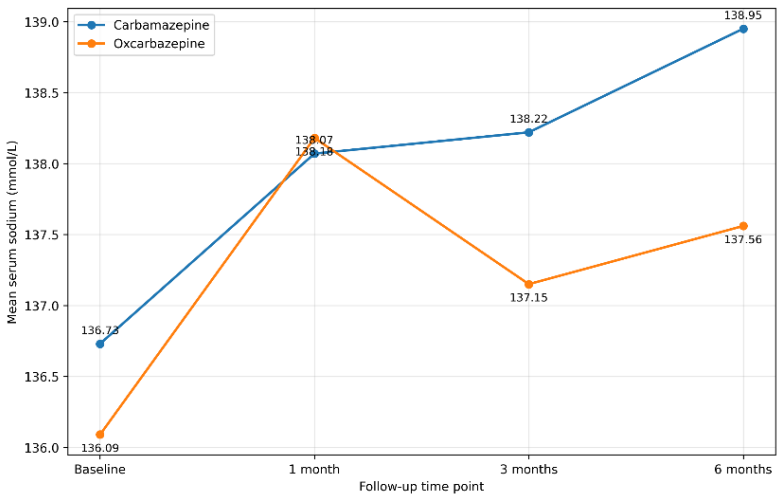

Figure 1: Mean serum sodium level over 6 months by treatment group.

Mean serum sodium increased from baseline in both treatment groups during follow-up. In the carbamazepine group, mean sodium increased by 1.34 mmol/l at 1 month, 1.49 mmol/l at 3 months, and 2.22 mmol/l at 6 months. In the oxcarbazepine group, mean sodium increased by 2.09 mmol/l at 1 month, 1.06 mmol/l at 3 months, and 1.47 mmol/l at 6 months. The between-group difference in mean change favored carbamazepine at 3 months and 6 months, with differences of 0.43 mmol/l and 0.75 mmol/l, respectively (Table 5).

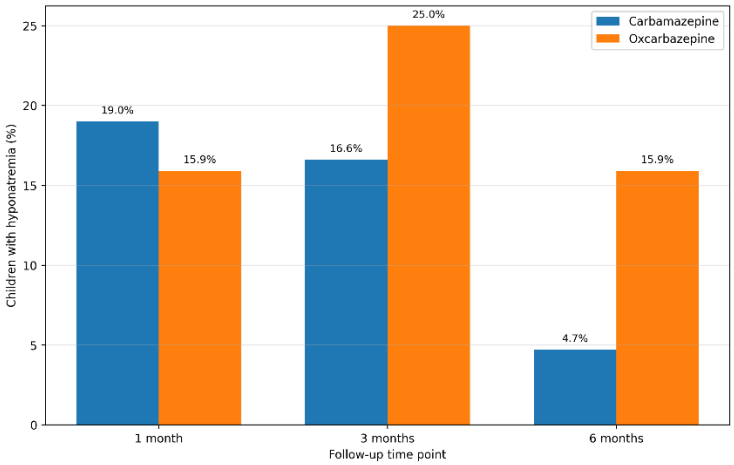

Figure 2: Proportion of children developing hyponatremia during follow-up.

Mean serum sodium levels were similar between treatment groups at baseline and 1 month. From 3 months onward, the oxcarbazepine group showed lower mean serum sodium than the carbamazepine group. At 6 months, mean serum sodium was 138.95 mmol/l in the carbamazepine group compared with 137.56 mmol/l in the oxcarbazepine group, consistent with a significant between-group difference (Figure 1).

The proportion of children with hyponatremia varied across follow-up visits. At 1 month, hyponatremia was slightly more frequent in the carbamazepine group, 19.0% versus 15.9%. At 3 months and 6 months, hyponatremia was more frequent in the oxcarbazepine group, 25.0% versus 16.7% at 3 months and 15.9% versus 4.8% at 6 months. However, these between-group differences were not statistically significant (Figure 2).

DISCUSSION

This randomized controlled trial compared serum sodium changes among children with focal epilepsy receiving carbamazepine or oxcarbazepine therapy over 6 months. The principal finding was that mean serum sodium was significantly lower in the oxcarbazepine group than in the carbamazepine group at 3 months and 6 months, despite comparable baseline and 1-month values. At 6 months, mean serum sodium was 137.56±2.54 mmol/l in the oxcarbazepine group compared with 138.95±2.20 mmol/l in the carbamazepine group, with a mean difference of 1.39 mmol/l. However, categorical hyponatremia was not significantly different between groups, although it was numerically higher with oxcarbazepine at 3 months and 6 months. The lower mean sodium observed with oxcarbazepine is consistent with previous evidence that oxcarbazepine has a stronger association with sodium reduction than carbamazepine. In a pediatric study of 197 epileptic children receiving carbamazepine and/or oxcarbazepine, Lee et al reported an overall hyponatremia prevalence of 20.8%, with rates of 17.9% in carbamazepine-treated children and 22.6% in oxcarbazepine-treated children.[11] This pattern is directionally similar to the present trial, where hyponatremia was more frequent with oxcarbazepine at later follow-up. A larger cohort study by Berghuis et al also reported higher hyponatremia rates with oxcarbazepine than carbamazepine, 46% versus 26%, and higher severe hyponatremia rates, 22% versus 7%.[7] The absolute rates in the present study were lower, which may reflect the pediatric population, short follow-up duration, monotherapy setting, exclusion of children with abnormal baseline sodium, and limited sample size. The finding that hyponatremia was numerically but not statistically higher with oxcarbazepine requires cautious interpretation. At 6 months, hyponatremia occurred in 15.9% of children receiving oxcarbazepine and 4.8% receiving carbamazepine, corresponding to a risk ratio of 3.34, but the confidence interval was wide and the p value was not significant. This suggests a possible clinically relevant signal that the study was not adequately powered to confirm as a categorical endpoint. The 3-month oxcarbazepine hyponatremia rate of 25.0% was close to the 23.3% rate reported in the control arm of the SCHO Trial among children receiving oxcarbazepine monotherapy without sodium supplementation.[10] This supports the external plausibility of the observed pediatric oxcarbazepine-associated hyponatremia frequency. Severe and symptomatic hyponatremia were uncommon in this trial. Only one child in the oxcarbazepine group developed severe hyponatremia, and no symptomatic hyponatremia, hospitalization, or treatment-attributable death occurred. This is compatible with pediatric data suggesting that clinically severe sodium disturbance is less frequent in children than in older populations. Yamamoto et al, in a large real-world epilepsy cohort, found lower moderate-severe hyponatremia incidence in pediatric patients than in adults and older adults.[9] However, the absence of symptoms should not be interpreted as absence of clinical importance. Berghuis et al showed that carbamazepine- and oxcarbazepine-induced hyponatremia may be associated with adverse symptoms, including dizziness, tiredness, instability, and diplopia.[8] Therefore, biochemical monitoring remains relevant even when

children appear clinically well. Mean serum sodium did not decline from baseline in either treatment group. Instead, both groups showed higher mean sodium over time, but the increase was smaller with oxcarbazepine at later follow-up. This finding should be described precisely. The trial does not demonstrate a mean sodium fall from baseline; rather, it demonstrates lower follow-up sodium values with oxcarbazepine compared with carbamazepine. Longer treatment duration may be important, as Čiauškaitė et al observed that longer oxcarbazepine exposure was associated with lower sodium levels and higher hyponatremia risk.[6] Overall adverse effects were not significantly different between the two groups. This finding is consistent with pediatric evidence showing generally acceptable oxcarbazepine tolerability. Geng and Wang found comparable adverse-event rates between oxcarbazepine and other antiepileptic drugs in children, while Liu et al reported no significant difference in total adverse reactions between levetiracetam and oxcarbazepine.[4,5] Similarly, a pharmacovigilance study by Lee et al found no significant difference in overall adverse drug reaction incidence between carbamazepine and oxcarbazepine.[12] The significantly higher anorexia rate with oxcarbazepine in the present trial should be interpreted cautiously, as this specific signal has not been consistently emphasized in larger pediatric safety studies.

Limitations

The study has limitations. It was single-centre, open-label, and relatively small, with per-protocol rather than intention-to-treat analysis. Follow-up was limited to 6 months, and raw change-score modelling was not available. Nevertheless, the randomized design, pediatric focal epilepsy population, and serial sodium monitoring provide useful evidence from a South Asian clinical setting where comparative pediatric data remain limited.

CONCLUSION

In this randomized controlled trial of children with focal epilepsy, oxcarbazepine therapy was associated with significantly lower mean serum sodium levels than carbamazepine at 3 months and 6 months of follow-up. However, the proportion of children developing hyponatremia was not significantly different between groups, although it was numerically higher with oxcarbazepine at later follow-up. Severe hyponatremia was rare, symptomatic hyponatremia was not observed, and both drugs were generally well tolerated. These findings suggest that serum sodium monitoring is clinically relevant in children receiving oxcarbazepine, even when overt symptoms are absent.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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