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Neurocognitive Trajectories Following Monthly Pulse IV Methylprednisolone in Children with Epileptic Encephalopathy

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Abstract

Background: Pediatric epileptic encephalopathies (EE) are characterized by drug-resistant seizures and cognitive regression. Corticosteroid therapies such as intravenous methylprednisolone (IVMP) have shown variable efficacy. This study evaluates the clinical, neurocognitive, and electrophysiological outcomes of IVMP in children with EE.

Methods: A retrospective cohort of 25 children diagnosed with EE (Lennox-Gastaut Syndrome [LGS], CSWS, West Syndrome) received IVMP pulse therapy. Outcomes assessed included seizure response, EEG improvement, cognitive shift, and treatment tolerability. Univariate logistic regression explored predictors of cognitive improvement.

Results: Seizure reduction ≥50% was observed in 64.0% overall, significantly higher in CSWS (100%) than LGS (30.8%, $P = 0.001$). EEG improvement occurred in 60.0%, also favoring CSWS (81.8% vs. 38.5%, $P = 0.047$). Cognitive improvement was noted in only 16.0% of patients, with no statistically significant predictors. Adverse events were mild and reported in 28.0%.

Conclusions: IVMP therapy may effectively reduce seizures and EEG burden in children with EE, particularly in CSWS. However, cognitive gains remain limited, emphasizing the need for adjunctive neurorehabilitative strategies. IVMP appears to be safe and well-tolerated.

Keywords: Epileptic encephalopathy, methylprednisolone, CSWS, EEG, cognitive outcomes

Introduction

Epileptic encephalopathy (EE) encompasses a group of severe childhood-onset epilepsy syndromes in which epileptiform activity itself contributes to cognitive and behavioral regression, beyond what can be attributed to underlying etiology alone. The clinical spectrum includes Lennox–Gastaut syndrome (LGS), continuous spike-and-wave during sleep (CSWS), and West syndrome each characterized by drug-resistant seizures, developmental stagnation or regression, and electroencephalographic (EEG) abnormalities such as diffuse spike-wave discharges.[1]

These syndromes represent some of the most neurologically disabling conditions in pediatric populations, with profound consequences for language, cognition, and adaptive functioning. Epidemiological estimates suggest a combined prevalence of 0.5 to 1.5 per 1,000 children, but in low- and middle-income countries (LMICs), underdiagnosis and resource limitations may conceal the true burden.[2]

Mounting evidence supports the concept that epileptic encephalopathies are not only seizure disorders, but also neurodevelopmental disorders mediated in part by ongoing inflammatory and immune dysregulation. Interictal epileptiform discharges (IEDs), particularly during non-REM sleep, have been shown to disrupt cognitive processing and lead to downstream network dysfunction.[3] In syndromes such as CSWS and Landau–Kleffner syndrome (LKS), spike-wave discharges during slow-wave sleep are strongly associated with deteriorations in language, behavior, and executive functioning.[4] Several authors now posit that inflammatory mediators may exacerbate this cortical hyperexcitability, contributing to neurocognitive decline even in the absence of clinical seizures.[5,6]

This pathophysiological model has catalyzed interest in immunomodulatory interventions for epileptic encephalopathy, especially corticosteroids. Pulse intravenous methylprednisolone (IVMP) has emerged as a candidate therapy due to its anti-inflammatory and neuroprotective properties, with applications across CSWS, LKS, and other variants.[7] In a recent prospective trial, IVMP led to significant EEG improvements and seizure reduction in children with EE, although cognitive outcomes were less robust, highlighting the need for further syndrome-specific investigation.[8] The literature suggests that EEG normalization is associated with improved neurocognitive function in some cases, but the variability of response and the precise predictors of improvement remain poorly characterized.[9]

Despite these insights, data from LMICs, and particularly from South Asia, remain scarce. In these settings, children with EE often face diagnostic delays, fragmented care, limited access to EEG monitoring, and inconsistent treatment protocols. A recent study from a tertiary care center in Dhaka, Bangladesh, described structural barriers to early EEG-based diagnosis and emphasized the absence of any longitudinal cognitive follow-up despite corticosteroid therapy.[10] Even globally, most existing studies have focused on seizure outcomes rather than long-term neurocognitive trajectories, and nearly all have been conducted in high-income countries with relatively homogeneous populations.[1,2] This creates a critical evidence gap in understanding the real-world effectiveness of IVMP in heterogeneous, resource-limited populations.

Addressing this gap, the current study evaluates the neurocognitive and electrophysiological outcomes following monthly pulse IVMP therapy in a prospective cohort of Bangladeshi children diagnosed with epileptic encephalopathy.

Methods

This was a prospective, observational cohort study conducted from January 2020 to June 2021 at the Department of Paediatric Neurology and the Institute of Paediatric Neurodisorder and Autism (IPNA), Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh. Ethical approval was obtained from the BSMMU Institutional Review Board, and informed written consent was collected from parents or legal guardians of all participants. Children under 12 years with a clinical diagnosis of epileptic encephalopathy (EE) were enrolled consecutively. Diagnosis followed ILAE electroclinical criteria and required failure of at least two appropriate antiepileptic drugs (AEDs). Exclusion criteria included critical illness, status epilepticus, or IV methylprednisolone (IVMP) use in the past six months. Of 32 eligible children, 7 were

lost to follow-up. The final analysis included 25 children who completed all four planned IVMP cycles. Baseline EEG was performed using a 21-electrode setup (10–20 system) capturing both wake and sleep states, analyzed using SystemPlus Evolution Micromed software. EEG spike index was calculated as the percentage of sleep epochs showing epileptiform discharges. Syndrome classification (LGS, CSWS, West syndrome) was based on EEG and clinical features. All patients received IVMP at 30 mg/kg/day (max 1 g/day) for five days, repeated monthly for four cycles, while continuing existing AEDs (typically sodium valproate, levetiracetam, and/or clobazam). Cognitive function was assessed before and one month after the final IVMP cycle using standardized developmental tools. Data were analyzed using SPSS v26. Categorical variables were expressed as frequencies and percentages; continuous variables as mean ± SD or median (IQR). Fisher's exact test and independent *t*-tests were used for between-group comparisons. Univariate logistic regression identified predictors of cognitive improvement, EEG response, and seizure reduction. Variables with $P < 0.10$ entered multivariate logistic regression. Statistical significance was set at $P < 0.05$.

Results

A total of 25 children with epileptic encephalopathy (EE) were included in the final analysis, comprising 13 cases of Lennox-Gastaut syndrome (LGS), 11 with continuous spike-and-wave during sleep (CSWS), and one with West syndrome. The mean age at initiation of IVMP therapy was 5.96 ± 3.01 years, with LGS patients being slightly older (6.8 ± 2.9 years) than those with CSWS (4.9 ± 2.8 years). A majority of the cohort were male (76.0%), with similar gender distribution across LGS (76.9%) and CSWS (72.7%). The mean age of seizure onset across the cohort was 1.8 ± 2.3 years, with earlier onset observed in the CSWS group (1.4 years) compared to LGS (2.1 years). Approximately 28.0% of the total cohort had seizure onset before one year of age, and 88.0% experienced multiple seizure types, which was universally present in LGS (100%). All children exhibited pre-existing cognitive impairment, while other developmental comorbidities were common. Motor impairment and speech difficulties were each reported in 44.0% of the cohort, with higher rates in LGS (53.8% and 46.2%, respectively). Visual and hearing impairments were less frequent, affecting 24.0% and 12.0% of children, respectively. Regarding etiology, structural brain abnormalities were observed in 36.0% of the children, more commonly among those with LGS (46.2%) than CSWS (18.2%). A significant proportion of cases (56.0%) had unknown or undetermined etiology, with this being most frequent in the CSWS group (72.7%). Neuroimaging was available for 18 patients, among whom half had abnormal findings. Cortical atrophy (24.0%) and ventricular dilatation (20.0%) were the most prevalent abnormalities. Other findings included cystic encephalomalacia (16.0%), corpus callosum agenesis or hypoplasia (8.0%), cerebellar atrophy (8.0%), and cerebral infarction (8.0%). Co-morbid neurodevelopmental disorders were identified in several cases. Cerebral palsy was present in 20.0% of the cohort—most notably in LGS (30.8%) and the single West syndrome patient. Attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) were reported in 8.0% and 4.0% of the cohort, respectively, with a slightly higher representation among CSWS cases [Table 1].

Following four monthly cycles of intravenous methylprednisolone (IVMP), cognitive improvement was observed in 4 out of 25 children (16.0%). The highest rate of improvement was noted in the single West syndrome case (100%), while cognitive gains were seen in 2 patients with CSWS (18.2%) and 1 with LGS (7.7%). However, the difference between LGS and CSWS in terms of cognitive response was not statistically significant ($P = 0.576$). Seizure response, defined as greater than 50% reduction in seizure frequency, was observed in 64.0% of the cohort (16 children). Notably, all children with CSWS

Table 1: Baseline characteristics of the study cohort (n = 25)

Characteristic	Overall (n = 25)	LGS (n = 13)	CSWS (n = 11)	West syndrome (n = 1)
Demographics				
Age at treatment (years), mean ± SD [range]	5.96 ± 3.01 [0.7–12]	6.8 ± 2.9 [2–12]	4.9 ± 2.8 [0.7–10]	0.8
Male, *n* (%)	19 (76.0%)	10 (76.9%)	8 (72.7%)	1 (100%)
Clinical features				
Age at seizure onset (years), mean ± SD	1.8 ± 2.3	2.1 ± 2.5	1.4 ± 2.0	0.3
Seizure onset <1 year, *n* (%)	7 (28.0%)	3 (23.1%)	3 (27.3%)	1 (100%)
Multiple seizure types, *n* (%)	22 (88.0%)	13 (100%)	8 (72.7%)	1 (100%)
Developmental status				
Pre-existing cognitive impairment, *n* (%)	25 (100%)	13 (100%)	11 (100%)	1 (100%)
Motor impairment, *n* (%)	11 (44.0%)	7 (53.8%)	3 (27.3%)	1 (100%)
Speech difficulty, *n* (%)	11 (44.0%)	6 (46.2%)	4 (36.4%)	1 (100%)
Visual impairment, *n* (%)	6 (24.0%)	4 (30.8%)	1 (9.1%)	1 (100%)
Hearing impairment, *n* (%)	3 (12.0%)	2 (15.4%)	0 (0%)	1 (100%)
Etiology				
Structural brain abnormality, *n* (%)	9 (36.0%)	6 (46.2%)	2 (18.2%)	1 (100%)
Genetic abnormality, *n* (%)	1 (4.0%)	0 (0%)	1 (9.1%)	0 (0%)
Metabolic abnormality, *n* (%)	1 (4.0%)	1 (7.7%)	0 (0%)	0 (0%)
Unknown/undetermined, *n* (%)	14 (56.0%)	6 (46.2%)	8 (72.7%)	0 (0%)
Neuroimaging findings (n = 18)				
Normal, *n* (%)	9 (36.0%)	4 (30.8%)	5 (45.5%)	0 (0%)
Abnormal, *n* (%)	9 (36.0%)	6 (46.2%)	2 (18.2%)	1 (100%)
Cortical atrophy	6 (24.0%)	4 (30.8%)	1 (9.1%)	1 (100%)
Ventricular dilatation	5 (20.0%)	3 (23.1%)	1 (9.1%)	1 (100%)
Cystic encephalomalacia	4 (16.0%)	3 (23.1%)	0 (0%)	1 (100%)
Corpus callosum agenesis/hypoplasia	2 (8.0%)	1 (7.7%)	0 (0%)	1 (100%)
Cerebellar atrophy	2 (8.0%)	1 (7.7%)	1 (9.1%)	0 (0%)
Cerebral infarction	2 (8.0%)	1 (7.7%)	1 (9.1%)	0 (0%)
Co-morbidities				
Cerebral palsy, *n* (%)	5 (20.0%)	4 (30.8%)	0 (0%)	1 (100%)
Attention deficit hyperactivity disorder, *n* (%)	2 (8.0%)	1 (7.7%)	1 (9.1%)	0 (0%)
Autism spectrum disorder, *n* (%)	1 (4.0%)	0 (0%)	1 (9.1%)	0 (0%)

(100%) and the West syndrome case (100%) responded to treatment, whereas only 4 out of 13 (30.8%) LGS patients met the responder criteria. This difference between LGS and CSWS was statistically significant (P = 0.001), indicating superior seizure control in CSWS. Electrophysiological improvement, defined as more than 50% reduction in spike-wave index on EEG, was recorded in 15 children (60.0%). EEG response was again highest among CSWS patients (81.8%)

and the West syndrome case (100%), compared to only 38.5% in LGS. The between-group difference in EEG improvement was statistically significant ($P = 0.047$), favoring CSWS. In terms of safety, 7 children (28.0%) experienced at least one adverse event during the treatment course. Adverse events were reported in 3 patients each from the LGS (23.1%) and CSWS (27.3%) groups, and in the sole West syndrome patient (100%), with no significant difference in overall adverse event rates ($P = 1.000$). Specific adverse effects included irritability (16.0%), hypertension (8.0%), and hypertrichosis (4.0%). No instances of hyperglycemia or significant weight gain were recorded [Table 2].

The distribution of cognitive function categories before and after IVMP therapy revealed limited shifts in neurocognitive status across the cohort. Out of 25 children, 2 (8.0%) were classified as cognitively "Normal" post-treatment, both of whom were in the "Normal" or "Borderline" categories at baseline. Six children (24.0%) were classified as "Borderline" post-treatment, including 1 who had been "Normal" and 5 who remained stable from their pre-treatment "Borderline" status. Among the 5 children with mild cognitive impairment at baseline, 4 remained within the same category, while 1 showed improvement to the "Borderline" level. The majority of children with moderate impairment (6 of 8) showed no change, and 2 of them deteriorated to the "Severe/Untestable" category. All 5 children with severe or untestable cognitive function at baseline remained unchanged following treatment. Overall, cognitive category remained unchanged in 21 of 25 patients (84.0%), with only 4 children (16.0%) demonstrating any upward shift in cognitive category post-IVMP. No instances of cognitive worsening were observed in children who began with "Normal," "Borderline," or "Mild Impairment" status [Table 3].

Univariate logistic regression was performed to identify potential predictors of cognitive improvement after IVMP therapy. While none of the predictors reached statistical significance, several trends were noted. Children with CSWS had higher odds of cognitive improvement compared to those with LGS (OR = 2.67, 95% CI: 0.21–34.45; $P = 0.461$), though this was not statistically significant. The single case of West syndrome was excluded from regression due to insufficient sample size. Among comorbidities, Cerebral Palsy showed a trend toward lower odds of improvement (OR = 0.32, 95%

Table 2: Neurocognitive and electrophysiological outcomes following IVMP therapy

Outcome measure	Overall ($n = 25$)	LGS ($n = 13$)	CSWS ($n = 11$)	West syndrome ($n = 1$)	*P*-value (LGS vs. CSWS)
Primary outcome					
Cognitive improvement, *n* (%)	4 (16.0%)	1 (7.7%)	2 (18.2%)	1 (100%)	0.576
Secondary outcomes					
Seizure responder (>50% reduction), *n* (%)	16 (64.0%)	4 (30.8%)	11 (100.0%)	1 (100%)	**0.001**
EEG improvement (>50% reduction in spike index), *n* (%)	15 (60.0%)	5 (38.5%)	9 (81.8%)	1 (100%)	**0.047**
Safety & Tolerability					
Any adverse event, *n* (%)	7 (28.0%)	3 (23.1%)	3 (27.3%)	1 (100%)	
Hypertension	2 (8.0%)	1 (7.7%)	0 (0%)	1 (100%)	
Irritability	4 (16.0%)	2 (15.4%)	1 (9.1%)	1 (100%)	1.000
Hypertrichosis	1 (4.0%)	0 (0%)	1 (9.1%)	0 (0%)	
Hyperglycemia	0 (0%)	0 (0%)	0 (0%)	0 (0%)	
Significant weight gain	0 (0%)	0 (0%)	0 (0%)	0 (0%)	

CI: 0.03–3.58; P = 0.357). Neither ADHD (OR = 1.60, P = 0.713) nor ASD (OR = 0.98, P = 0.990) showed meaningful associations with cognitive outcomes. Regarding concomitant AEDs, use of Clobazam had the highest observed odds of cognitive improvement (OR = 3.25, 95% CI: 0.29–36.12; P = 0.337), though confidence intervals were wide. Sodium valproate (OR = 1.85) and levetiracetam (OR = 0.52) did not significantly predict outcomes. Notably, electrophysiological markers such as EEG improvement (OR = 4.20, 95% CI: 0.40–44.56; P = 0.235) and seizure response (OR = 3.00, P = 0.373) showed numerically higher odds for cognitive gain, suggesting potential predictive value, though not statistically confirmed in this analysis. Etiological factors such as structural brain abnormalities (OR = 0.44) and unknown etiology (OR = 1.75) also did not yield statistically significant associations [Table 4].

Table 3: Shift in cognitive assessment categories pre- and post-IVMP therapy

Post-treatment	Normal	Borderline	Mild impairment	Moderate impairment	Severe/ Untestable	Total Pre-Tx
Normal	1	0	0	0	0	1
Borderline	1	5	0	0	0	6
Mild impairment	0	1	4	0	0	5
Moderate impairment	0	0	0	6	2	8
Severe/Untestable	0	0	0	0	5	5
Total Post-Tx	**2**	**6**	**4**	**6**	**7**	**25**

Table 4: Univariate logistic regression analysis of predictors for cognitive improvement

Predictor	Odds ratio (OR)	95% Confidence interval	*P*-value
Syndrome type (Ref: LGS)			
CSWS	2.67	0.21 – 34.45	0.461
West syndrome*	–	–	–
Co-morbidities			
Cerebral palsy (Yes vs. No)	0.32	0.03 – 3.58	0.357
ADHD (Yes vs. No)	1.60	0.13 – 19.31	0.713
ASD (Yes vs. No)	0.98	0.06 – 16.58	0.990
Concomitant AEDs			
Sodium Valproate (Yes vs. No)	1.85	0.16 – 21.79	0.628
Levetiracetam (Yes vs. No)	0.52	0.05 – 5.28	0.577
Clobazam (Yes vs. No)	3.25	0.29 – 36.12	0.337
Electrophysiological response			
EEG improvement (Yes vs. No)	4.20	0.40 – 44.56	0.235
Seizure response (Yes vs. No)	3.00	0.27 – 33.66	0.373
Etiology			
Structural abnormality (Yes vs. No)	0.44	0.04 – 4.97	0.510
Unknown etiology (Yes vs. No)	1.75	0.17 – 18.40	0.642

*Note: The West syndrome case (n = 1) was excluded from this regression analysis due to insufficient sample size for the model. Ref = Reference category.

Discussion

This prospective cohort study evaluated the cognitive and electrophysiological outcomes following monthly intravenous pulse methylprednisolone (IVMP) therapy in children with epileptic encephalopathy (EE), primarily Lennox-Gastaut syndrome (LGS), continuous spike-and-wave during sleep (CSWS), and a single case of West syndrome. The results offer important insights into the real-world clinical response to IVMP in a low-resource South Asian setting.

The demographic profile of our cohort predominantly male, with a mean treatment age of approximately 6 years and seizure onset around 1.8 years—is consistent with previously described pediatric EE populations.[11,12] Most children presented with multiple seizure types and developmental delay, underscoring the typical complexity of these syndromes.[13] Notably, over half the cases had undetermined etiology, highlighting a persistent diagnostic gap in low- and middle-income countries (LMICs), a pattern similarly observed in studies from Nepal and India.[11,12] Structural brain abnormalities—especially cortical atrophy and ventricular dilatation were the most common identifiable etiologies, in line with other Asian pediatric series.[12,14]

The primary cognitive outcome showed that only 16% of the cohort demonstrated measurable improvement after four cycles of IVMP. This modest response is consistent with prior investigations that showed cognitive gains in a minority of patients, often in relation to specific syndrome types or early treatment initiation.[8,15,16] Our findings of higher improvement rates in CSWS and West syndrome compared to LGS mirror patterns reported in both observational and controlled studies, suggesting syndrome-specific responsiveness to corticosteroid therapy.[17,18] However, these differences were not statistically significant in our cohort, likely due to the small sample size and heterogeneity.

Electrophysiological outcomes were more favorable: 60% of children showed ≥50% reduction in spike-wave activity, and 64% experienced significant seizure reduction. These findings echo those reported in other IVMP-treated cohorts, where seizure and EEG improvements were frequently observed despite limited cognitive changes.[8,15–17] Our study notably found that CSWS had significantly better seizure and EEG outcomes than LGS ($P = 0.001$ and $P = 0.047$, respectively), reinforcing prior evidence that CSWS is particularly amenable to corticosteroid therapy.[18,19] This may be due to the presumed immune-mediated pathophysiology and non-ictal network dysfunction that underlie CSWS, making it more steroid-responsive than structural or genetically-driven epileptic encephalopathies like LGS.[5,17]

Despite electrophysiological and clinical gains, cognitive trajectories remained largely static. In 84% of children, cognitive categorization did not shift, and no patient with severe baseline impairment improved. These findings are echoed in the work of Wright and Wood, who observed similar cognitive stability in children with immune-mediated EE despite immunomodulatory treatments.[20] The results also align with Becker and Kaindl's systematic review, which concluded that while corticosteroids often improve seizures and EEG, cognitive changes are variable and rarely robust.[5] It is possible that neurodevelopmental damage in many of these children is already entrenched by the time of treatment, particularly in resource-limited settings where delays in diagnosis and therapy are common.

Our regression analysis did not identify any statistically significant predictors of cognitive improvement. However, the directionality of trends greater odds of improvement in children with CSWS, EEG improvement, and seizure response is biologically and clinically plausible, and consistent with prior findings.[5,16,17] Similarly, clobazam use was associated with the highest odds of cognitive gain among antiepileptics, resonating

with comparative studies where clobazam was seen as an effective adjunct in certain EE phenotypes.[19] Conversely, the presence of structural brain abnormalities and comorbid cerebral palsy trended toward poorer cognitive outcomes, aligning with previous reports where neurodevelopmental comorbidities often signaled limited reversibility.[11,21]

IVMP therapy was generally well tolerated. Adverse events occurred in 28% of patients, with irritability and hypertension being the most reported effects findings consistent with safety profiles reported across studies.[8,15,22] Importantly, no serious or life-threatening complications were noted, reinforcing the feasibility of IVMP use even in low-resource environments.

This study adds to the growing literature supporting the use of pulse corticosteroids in selected EE syndromes. However, it also underscores the limitations of IVMP in inducing significant short-term cognitive improvement, particularly in children with severe baseline impairment or complex structural comorbidities. Future studies should explore the role of early intervention, combination therapy strategies, and longer follow-up periods to better delineate the long-term cognitive impact.

Limitations of the Study

The study was conducted in a single hospital with a small sample size. So, the results may not represent the whole community.

Conclusion

This study demonstrates that intravenous methylprednisolone (IVMP) pulse therapy may offer clinical benefits in pediatric epileptic encephalopathies (EE), particularly in reducing seizure frequency and improving EEG abnormalities, most notably in children with Continuous Spike-and-Wave during Sleep (CSWS). However, cognitive improvement was modest and limited to a small proportion of patients, with no statistically significant predictors identified. While the treatment was generally well tolerated, the lack of substantial neurocognitive gain underscores the need for more targeted and multimodal interventions. These findings reinforce IVMP's role as a seizure-modulating strategy rather than a robust cognitive enhancer in pediatric EE. Further multicentric, prospective studies with larger sample sizes and longer follow-up are needed to better delineate the subset of patients who may benefit most, and to explore synergistic therapeutic avenues for neurocognitive recovery.

Ethical Approval

The study was approved by the Institutional Ethics Committee.

References

1. Raga S, Specchio N, Rheims S, Wilmshurst JM. Developmental and epileptic encephalopathies: recognition and approaches to care. Epileptic Disord 2021 Feb 1; 23(1):40–52.
2. Raga SV, Wilmshurst JM. Epileptic spasms: evidence for oral corticosteroids and implications for low and middle income countries. Seizure 2018 Jul;59:90–8.
3. Van Bogaert P. Epileptic encephalopathy with continuous spike-waves during slow-wave sleep including Landau-Kleffner syndrome. Handb Clin Neurol 2013;111:635–40.
4. Kotagal P. Current Status of Treatments for Children with Electrical Status in Slow-Wave Sleep (ESES/CSWS). Epilepsy Curr [Internet]. 2017 [cited 2025 Sep 1];17(4):214–6. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5716110/
5. Becker LL, Kaindl AM. Corticosteroids in childhood epilepsies: a systematic review. Front Neurol [Internet]. 2023 Mar 10 [cited 2025 Sep 1];14. Available from: https://www.frontiersin.org/journals/neurology/articles/10.3389/fneur.2023.1142253/full
6. Xiaoyue H, Hongwei T, Jianbiao W, Jingbo M, Ying H. Exploratory analysis of high-dose corticosteroid therapy on epileptic encephalopathy with spike-and-wave activation in sleep. Front Pediatr 2024;12:1388008.

7. Jauhari P, Madaan P, Sirolia V, Sharma S, Chakrabarty B, Gulati S. Neurobehavioral deterioration associated with sleep-augmented epileptiform abnormalities: A steroid responsive state in children. Epilepsy Behav 2022 Apr;129:108505.
8. Rangarajan A, Mundlamuri RC, Kenchaiah R, Prathyusha PV, Viswanathan LG, Asranna A, et al. Efficacy of pulse intravenous methylprednisolone in epileptic encephalopathy: a randomised controlled trial. J Neurol Neurosurg Psychiatry 2022 Dec;93(12):1299–305.
9. Saraf UU, Asranna A, Menon RN, Mohan PM, Vp V, Radhakrishnan A, et al. Predictors of one-year language and seizure outcomes in children with epileptic encephalopathy with continuous spike-and-wave during sleep (CSWS). Seizure 2020 Oct;81:315–24.
10. Abath CB, Chandra Saha N, Hoque SA, Islam A, Chowdhury YS, Ara Begum MS, et al. Clinical characteristics of children with infantile epileptic spasms syndrome from a tertiary-care hospital in Dhaka, Bangladesh. Heliyon 2023 Mar;9(3):e14323.
11. Bhanudeep S, Madaan P, Sankhyan N, Saini L, Malhi P, Suthar R, et al. Long-term epilepsy control, motor function, cognition, sleep and quality of life in children with West syndrome. Epilepsy Res 2021;173: 106629.
12. Poudel P, Kafle SP, Pokharel R. Clinical profile and treatment outcome of epilepsy syndromes in children: a hospital-based study in Eastern Nepal. Epilepsia Open 2021;6(1):206–15.
13. Covanis A. Epileptic encephalopathies (including severe epilepsy syndromes). Epilepsia 2012;53:114–26.
14. Barbarrosa EP, de la Caridad Pérez Ferrer I, Tovani-Palone MR. West Syndrome: Clinical Characteristics, Therapeutics, Outcomes and Prognosis. Electron J Gen Med 2020;17(2):em190.
15. Pera MC, Randazzo G, Masnada S, Dontin SD, De Giorgis V, Balottin U, et al. Intravenous methylprednisolone pulse therapy for children with epileptic encephalopathy. Funct Neurol 2015;30(3):173–6.
16. Chatterjee A, Mundlamuri RC, Kenchaiah R, Asranna A, Nagappa M, Bindu PS, et al. Role of pulse methylprednisolone in epileptic encephalopathy: a retrospective observational analysis. Epilepsy Res 2021;173:106611.
17. Russo A, Mazzone S, Landolina L, Colucci R, Baccari F, Fetta A, et al. Efficacy and safety of pulse intravenous methylprednisolone in pediatric epileptic encephalopathies: timing and networks consideration. J Clin Med 2024;13(9):2497.
18. Gulsan-Ara-Zahan GKK, Anwar DS. Clinical and Neuro Electrophysiological Outcome of Intravenous Pulse Methylprednisolone Therapy in Children with Continuous Spike and Waves during Slow Sleep: A Self-Controlled Clinical Trial. J Pediatr Perinatol Child Health 2024;8:89–94.
19. van den Munckhof B, Arzimanoglou A, Perucca E, van Teeseling HC, Leijten FS, Braun KP, et al. Corticosteroids versus clobazam in epileptic encephalopathy with ESES: a European multicentre randomised controlled clinical trial (RESCUE ESES*). Trials 2020;21(1):957.
20. Wright SK, Wood AG. Neurodevelopmental outcomes in paediatric immune-mediated and autoimmune epileptic encephalopathy. Eur J Paediatr Neurol 2020;24:53–7.
21. Operto FF, Pastorino GM, Viggiano A, Dell'Isola GB, Dini G, Verrotti A, et al. Epilepsy and cognitive impairment in childhood and adolescence: a mini-review. Curr Neuropharmacol 2023;21(8):1646–65.
22. Korinthenberg R, Bast T, Haberlandt E, Stephani U, Strzelczyk A, Rücker G. Efficacy and safety of corticosteroids and ACTH in epileptic syndromes beyond infantile epileptic spasms syndrome (IESS): a systematic review and meta-analysis. Epilepsia 2024;65(5):1155–75.

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