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Variations of Patterns of Electroencephalogram in West syndrom

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Abstract

Background: West syndrome (WS) or Infantile epileptic spasms syndrome (IESS) is a potentially devastating form of developmental and epileptic encephalopathy of early childhood. Early diagnosis and initiation of prompt treatment are very important for better outcome. But it has many differential diagnoses, especially in atypical or subtle cases. EEG is essential to diagnose it. Initially, hypsarrhythmia was described as the prototypical EEG pattern associated with WS. However, other EEG patterns were also found to be associated with WS in subsequent studies. Recognition of those patterns is important for appropriate diagnosis and prompt initiation of specific anti-seizure medications. Moreover, some of the EEG patterns were reported to be related to specific underlying aetiologies. **Objective:** This study was undertaken to identify various interictal EEG patterns in WS and to examine their relationship with etiology. **Methodology:** It was a cross-sectional observational study done at the Paediatric Neurology unit of, Bangabandhu Sheikh Mujib Medical University, Dhaka from January 2013 to June 2015, Clinically, WS was diagnosed by epileptic spasms with psychomotor delay or regression. Those were elicited by detailed history and thorough physical examination. EEG, neuroimaging and other diagnostic tests were performed. Those findings were analyzed. **Results:** Among 57 children with WS, EEG showed multifocal epileptiform discharges in 22(38.5%), burst suppression pattern in 18(31.5%), asymmetrical hypsarrhythmia in 9(15.7%), classic hypsarrhythmia in 5(8.7%) and increased interhemispheric synchronization in 3(5.2%) cases. However, no significant association was found between a particular EEG pattern and underlying aetiology. **Conclusion:** This study reveals that interictal EEG of children with WS may show various EEG patterns other than classic hypsarrhythmia. *[Journal of National Institute of Neurosciences Bangladesh, July 2025;11(2):104-109]*

Keywords: West syndrome; Infantile epileptic spasms syndrome; EEG; hypsarrhythmia; Modified hypsarrhythmia

Introduction

West syndrome (WS) or infantile epileptic spasms syndrome (IESS) is a potentially devastating form of developmental and epileptic encephalopathy that typically begins in first 2 years of life. The estimated incidence of WS is 30/100 000 live-born infants, with some studies suggesting higher incidence rates with higher geographic latitudes. A population- based cohort showed that WS accounted for 10.0% of epilepsy that begin prior to 36 months. WS is notable due to its age-specific onset, strong association with developmental retardation (75.0% to 93.0% of affected children), high mortality rate (11.0% to 33.0%), resistance to conventional anti-seizure medication, and unique responsiveness to hormonal therapy or Vigabatrin[1]. WS classically refers to the triad of epileptic spasms, hypsarrhythmia on electroencephalogram (EEG) and developmental stagnation or regression. Epileptic spasms start between 1 and 24 (peak 3 and 12) months of age. Infants may have no antecedent history, or the antecedent history may reflect the underlying cause, for example, acquired structural brain or genetic abnormality. In some cases, infants with early infantile developmental and epileptic encephalopathy or other early onset epilepsy (usually with focal seizures) may evolve to have clinical and EEG features of IESS after 3

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to 4 months of age. Epileptic spasms are defined as brief tonic contractions of axial muscles, each typically lasting less than 3 seconds, which may be flexor, extensor, or mixed. Those usually occurs in series or clusters, with increasing prominence of the motor features through the cluster, often over a period of minutes (although clusters may last 30 min or longer) and were often seen on awakening. Those may be symmetric or asymmetric and may be subtle, with minor head nods, or eye or chin movements[2]. Identifying EEG patterns can be crucial, as delayed diagnosis increases the risk of poor outcomes. Other epileptic and non-epileptic disorders can mimic spasms, but EEG features help differentiate WS[3,4], . Hypsarrhythmia is the interictal EEG pattern most associated with WS. Gibbs and Gibbs described it as random, high-voltage slow waves with focal or multifocal epileptiform discharges and marked variability[5]. Variants include hypsarrhythmia with voltage asymmetry, inter-hemispheric synchrony, focal discharges, voltage attenuation, or minimal spike activity[6,7]. These variants often correlate with underlying brain abnormalities. Additional interictal EEG patterns include focal/multifocal spikes, abnormal rhythms, diffuse or focal slowing, paroxysmal bursts, slow spike-and-wave, or continuous spindling[8,9]. Aetiologies of WS vary globally. In developed countries, non-progressive prenatal lesions dominate[10,11]. While perinatal/postnatal insults are significant in developing nations[12].Common prenatal causes include cortical dysplasia's, neurocutaneous disorders, malformations, and metabolic issues. Perinatal factors like asphyxia, seizures, and sepsis are also common. Consequently, EEG patterns may differ from region to region. One developed country study found burst suppression in 51.0%, asymmetry in 38.0%, and inter-hemispheric synchronization in 13.0% of WS cases[10]. On the other hand, a developing country study showed asymmetrical hypsarrhythmia in 12.0%, burst suppression in 8.0%, and multifocal discharges in 7.0%. Certain EEG patterns have aetiological and prognostic implications. For example, WS with cerebral dysgenesis often shows burst suppression and hemihypsarrhythmia[9]. Asymmetrical interictal EEG is linked to symptomatic causes and poor outcomes[13,14]. The ILAE recommends listing EEG variants separately rather than grouping them as 'modified hypsarrhythmia'. Though traditionally hypsarrhythmia is the hallmark interictal pattern in WS, other patterns also occur. Recognizing these is vital for accurate diagnosis and early treatment. This study aims to identify interictal EEG abnormalities in WS and examine their relationship with clinical features and aetiology.

Methodology

Study Design and Setting: All the newly diagnosed children with WS, aged 3 to 24 months, attending at the Paediatric Neurology unit of, Bangabandhu Sheikh Mujib Medical University, Dhaka from January 2013 to June 2015, were included. It was a cross-sectional observational study. Clinically, WS was diagnosed by epileptic spasms with psychomotor delay or regression. Those were elicited by detailed history and thorough physical examination.

Study Population: Epileptic spasms were defined as brief tonic contractions of axial muscles, each typically lasting less than 3 seconds, which may be flexor, extensor, or mixed. Those usually occurred in series or clusters, with increasing prominence of the motor features through the cluster, often over a period of minutes (although clusters may last 30 min or longer) and were often seen on awakening. Those might be symmetric or asymmetric and may be subtle, with minor head nods, or eye or chin movements. Epileptic spasms were documented from the home video recording of the event by the cellular phone of the caregivers. A data collection sheet was developed containing serial number, demographic information, case history, examination findings, laboratory investigations and EEG findings. Detailed history (gender of the patient, age at onset of spasms, age at presentation, developmental history, history of consanguinity of marriage, family history of similar illness and prenatal, perinatal and postnatal history) was taken and thorough general physical examination, head circumference, neurological examination, dermatological examination for stigmata suggestive of neurocutaneous disorders such as tuberous sclerosis complex, ophthalmologic assessment, and examination for dysmorphic features were done. Developmental status was assessed by history and bedside clinical evaluation of milestones of development. Informed written consent was taken from the guardians of the children. Samples were taken purposively. Patients fulfilling the inclusion criteria were enrolled consecutively as study population.

EEG Test: EEG was done at electrophysiology department of the BSMMU. Interictal EEG recordings obtained with a 24-channel digital EEG machine, employing scalp electrodes placed according to the international 10-20 system, were studied. Recording was collected at least for 30 minutes and was reported by two independent neurologists by using longitudinal, transverse, average referential, common referential and other montages. The assessors commented on overall

EEG findings like normal or abnormal, EEG background like normal or abnormal and presence of the abnormal EEG patterns. When there was a difference in the EEG reporting, two assessors discussed, and consensus was reached.

Neuroimaging Studies: Neuroimaging studies like ultrasonogram, computed tomography or magnetic resonance imaging of the brain were done according to the age, affordability of the caregivers and associated neurological condition. In clinically suspected cases, TORCH antibody titres in blood were seen. If TORCH antibody titres were significant, more specific tests like CMV DNA in urine for congenital CMV infection were done. In selected cases metabolic screening tests like blood ammonia, serum lactate, arterial blood gas analysis, urine for ketone bodies, urine for organic acids, plasma amino acids and acylcarnitine, were done. A chromosomal study was carried out where clinically indicated like Down syndrome. Children with WS were categorized according to baseline clinical characteristics (gender, age of onset of spasms, age at presentation, gestational age, and consanguinity of marriages between parents, family history of similar illness, developmental status, underlying disease association), neuroimaging findings, aetiology or associated neurological disorders and EEG findings.

Statistical Analysis: Descriptive statistics was used to describe different clinical parameters and EEG patterns, and was expressed as rates, percentages etc. Then EEG patterns were related to different clinical parameters and aetiologies. Fisher's exact Probability test was used to test the difference.

Ethical Consideration: No intervention was done in this study. Ethical clearance was taken from "Institutional review board" of Bangabandhu Sheikh Mujib Medical University, Dhaka (IRB 2014/994)

Results

A total of 75 children with suspected WS attended our department during the study period and 57 fulfilled the eligibility criteria. Clinical profile of the study population is shown in the table 1.

Age at onset of epileptic spasms and age at presentation to our centre are shown in the table 2. Most of the children had onset of epileptic spasms at the age of 3 to 5 months. Majority of the children presented to our centre between 6 to 11 months of age.

In this study, all the children were symptomatic WS. Perinatal injury was the major cause of WS. Perinatal asphyxia was the commonest aetiology among the perinatal injury (Figure 1). Prematurity (3), neonatal sepsis (3) and neonatal jaundice were other perinatal aetiologies. Prenatal causes included congenital Cytomegalovirus (CMV) infection (1) and inborn error of metabolism (2).

Table 1: Clinical profile of the children with WS

Characteristic	No. of the patients (Percent)
Gender (Male)	41 (72.0%)
Consanguinity of marriage between parents	6 (10.5%)
Family history of epilepsy	0
History of premature birth (< 37 weeks of gestation)	3(5.3%)
Mode of delivery (Spontaneous vaginal delivery)	50(87.7%)
Microcephaly	48(84.2 %)
Developmental delay	57 (100.0%)

Table 2: Age distribution according to the onset of Epileptic spasms and presentation

Age Group	Age at onset of Epileptic spasms	Age at Presentation to us
3 to 5 months	25	8
6 to 8 months	16	13
9 to 11 months	11	13
12 to 14 months	2	7
15 to 17 months	2	6
18 to 20 months	1	6
21 to 23 months	0	4

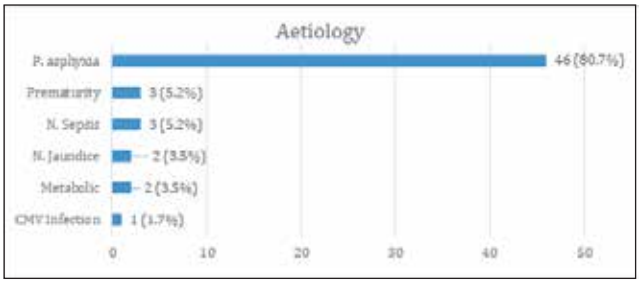

Figure I: Etiology or associated neurological disorders with West Syndrome.

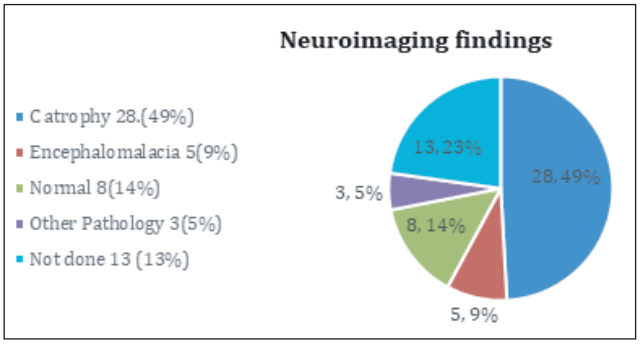

Figure II: Pie chart showing distribution of neuroimaging patterns in WS.

Table 3. Causes of Death of Study Population (n=100)

Cause of Death	Aetiology of WS (n, total cases in each group)					
	P. asphyxia (n=46)	CMV infection (n=1)	Prematurity (n=3)	Metabolic (n=2)	N. sepsis (n=3)	N. Jaundice (n=2)
Classic hypsarrhythmia (5)	5	0	0	0	0	0
Burst suppression (18)	13	0	1	1	2	1
Interhemispheric synchronization (3)	3	0	0	0	0	0
Multifocal epileptiform discharges (22)	16	1	2	1	1	1
Asymmetrical hypsarrhythmia (9)	9	0	0	0	0	0

P. asphyxia= Perinata asphyxia; N. sepsis= Neonatal sepsis, ; N. Jaundice=Neonatal Jaundice ,CMV =Cytomegalo virus.

Neuroimaging could be done in 44 (77.1%) children (Figure 2). Cerebral atrophy was the predominant finding (28). Other children had encephalomalacia (5) and other findings (3).

All the patients had abnormal interictal EEG (Table 3). Background was abnormal in all. Multifocal epileptiform discharges were the predominant abnormality (22). Classic hypsarrhythmia was found in only 5 patients. Burst suppression (18) and asymmetrical hypsarrhythmia (9) were present in other patients.

Abnormal EEG patterns were investigated for potential association with underlying aetiologies. But no statistically significant association was found.

Discussion

This study was undertaken to find out variations of EEG patterns in WS in Bangladesh. Baseline clinical characteristics, aetiology and neuroimaging findings were also studied. Efforts were made to see whether there was any relation with above mentioned parameters and EEG findings. In this study, WS were seen more frequently in males 41(72.0%). Fifty-four (94.7%) children were term and 3 (5.3%) were preterm. Six (10.5%) children with WS had history of consanguinity of marriage between parents. There was no family history of epilepsy in our cases. Genetic causes have been implicated with WS in various studies[15]. But detailed genetic study could not be done due to limitation in resources. This observation is supported by other studies from developing countries[16]. Most of the WS had the onset of epileptic spasms at the age 3 to 5 months. Majority of the WS presented to us at the age of 6 to 11 months. It was like the findings of the other studies[17]. All the WS were symptomatic in this study. Most common cause was perinatal asphyxia, seen in 46 patients (80.7%). Perinatal injury was also predominant aetiology in other developing countries Western studies found neuronal migration disorders (focal cortical dysplasia's, lissencephaly, hemimegaloencephaly and pachygyria), neurocutaneous and metabolic disorders as common causes of symptomatic group[19,11,12], This discrepancy may be due to the higher incidence of perinatal insults in Bangladesh due to various reasons.

Moreover, appropriate neuroimaging (e.g. MRI) studies, detailed metabolic work up and genetic investigations could not be done in every case due to resource limitation. All the patients have abnormal interictal EEG patterns. Classic hypsarrhythmia (CH) was found in 5(8.7%) cases. Hypsarrhythmia with burst suppression pattern was found in 18 (31.5 %), hypsarrhythmia with increased interhemispheric synchronization in 3 (5.0%), multifocal epileptiform discharges in 22 (38.5 %), and asymmetrical hypsarrhythmia in 9 (15.7 %). The prototypical pattern of CH reported to occur in from 7% to 75% of patients with WS[20,21,22]. This wide variation was due to differences in inclusion criteria, age at presentation, underlying associated diseases, duration of the recording, and clinical state of the patient (sleep or awake). Shortly after the first description of hypsarrhythmia by Gibbs and Gibb[5], Bower et al.[19] showed that this "classic" appearance had not been found in all patients with WS. Even, Gibbs and Gibbs also noted the variant of hypsarrhythmia with paucity of sharp and spike activity in some cases.[5] Gastaut et al8 classified hypsarrhythmia into typical and atypical, subclassified atypical into five main categories, and found that typical hypsarrhythmia comprised 63.0% of their sample. Hoefer et al[23] described hypsarrhythmia with burst suppression pattern. Ohtahara[24] delineated the pattern of asymmetric hypsarrhythmia[3]. In 1984, Hrachovy[25] and his colleagues reviewed multiple 24-hour EEG-video monitoring studies in 67 patients with WS and identified variations or modifications of the pattern as originally described. These patterns were referred as variants of hypsarrhythmia. They did not

show percentages of various patterns. Alva-Moncayo et al[7] confirmed the classification of Hrachovy et al[22] and provided the relative frequencies of those variants in 100 cases. Hypsarrhythmia with increased interhemispheric synchrony occurred most (35.0%), followed by hypsarrhythmia with a consistent focus of abnormal discharge (26.0%), asymmetric hypsarrhythmia (12.0%), hypsarrhythmia with episodes of voltage attenuation (11.0%), and hypsarrhythmia with high voltage with little spike or sharp wave activity (7.0%), Kramer et al[10] found burst suppression in 51.0% cases, asymmetry in 38.0% cases and interhemispheric synchronization in 13.0% cases. In this study significant number of patients had multifocal independent spike discharges. These observations are in line with the results of Watanabe et al[9] that multifocal spikes might evolve into hypsarrhythmia with long duration of untreated spasms. Gaily et al[14] also showed that multifocal independent spikes with normal or nearly normal background appeared to be another characteristic of WS with recent onset (in 44.0% of cases).

The potential association of each of EEG patterns with underlying aetiology was investigated. But no statistically significant association was found. Several studies showed that some EEG patterns have distinctive aetiological and prognostic implications, Watanabe et al[9] described that WS with cerebral dysgenesis has burst suppression pattern and hemi hypsarrhythmia. Asymmetrical interictal EEG is significantly associated with symptomatic aetiology and poor outcome[13,14]. This difference may be due to small sample size and difference in underlying associated diseases.

Conclusion

The study revealed that EEG can vary widely in WS. In addition to classic hypsarrhythmia, other patterns may be found frequently. Recognition of this variation in EEG is crucial for the early and appropriate diagnosis WS. However, no association between the EEG patterns and the other clinical characteristics or etiology was found. Larger prospective study with details investigations can find out some clinically important association.

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Data Availability
Any inquiries regarding supporting data availability of this study should be directed to the corresponding author and are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate
Ethical approval for the study was obtained from the Institutional Review Board. As this was a prospective study the written informed consent was obtained from all study participants. All methods were performed in accordance with the relevant guidelines and regulations.

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References

1. Vigevano F, Fusco L, Pachatz C. Neurophysiology of spasms. Brain and Development. 2001 Nov 1;23(7):467-72.
2. Zuberi SM, Wirrell E, Yozawitz E, Wilmshurst JM, Specchio N, Riney K, Pressler R, Auvin S, Samia P, Hirsch E, Galicchio S. ILAE classification and definition of epilepsy syndromes with onset in neonates and infants: Position statement by the ILAE Task Force on Nosology and Definitions. Epilepsia. 2022 Jun;63(6):1349-97.
3. Ohtahara S, Ohtsuka Y, Yamatogi Y, Oka E. The early-infantile

epileptic encephalopathy with suppression-burst: developmental aspects. Brain and Development. 1987 Jan 1;9(4):371-6.
4. Caraballo RH, Ruggieri V, Gonzalez G, Cersosimo R, Gamboni B, Rey A, Poveda JC, Dalla Bernardina B. Infantile spams without hypsarrhythmia: a study of 16 cases. Seizure. 2011 Apr 1;20(3):197-202.
5. Gibbs FA. Atlas of Electroence-phalography: Vol 2. Epilepsy.. 1952:210-25.
6. Hrachovy RA, Frost Jr JD, Kellaway P. Hypsarrhythmia: variations on the theme. Epilepsia. 1984 Jun;25(3):317-25.
7. Alva-Moncayo E, Diaz-Leal MC, Olmos-Garcia de Alba G. Electroencephalographic discoveries in children with infantile massive spasms in Mexico. Revista de Neurologia. 2002 May 1;34(10):928-32.
8. Gastaut H. Historique. L'Encephalopathie myocolonique infantile avec hypsarythmie (synrome de West). 1964:2-14.
9. Watanabe K, Negoro T, Aso K, Matsumoto A. Reappraisal of interictal electroencephalograms in infantile spasms. Epilepsia. 1993 Jul;34(4):679-85.
10. Kramer U, Sue WC, Mikati MA. Hypsarrhythmia: frequency of variant patterns and correlation with etiology and outcome. Neurology. 1997 Jan;48(1):197-203.
11. Osborne JP, Edwards SW, Dietrich Alber F, Hancock E, Johnson AL, Kennedy CR, Likeman M, Lux AL, Mackay M, Mallick A, Newton RW. The underlying etiology of infantile spasms (West syndrome): Information from the International Collaborative Infantile Spasms Study (ICISS). Epilepsia. 2019 Sep;60(9):1861-9.
12. Zhu L, Xia Y, Ding H, Zhang T, Li J, Li B. Infantile epileptic spasms syndrome: an etiologic study of 361 patients with infantile epileptic spasms syndrome. Frontiers in Pediatrics. 2025 Jan 9;12:1522079.
13. Drury I, Beydoun A, Garofalo EA, Henry TR. Asymmetric hypsarrhythmia: clinical electroencephalographic and radiological findings. Epilepsia. 1995 Jan;36(1):41-7.
14. Gaily E, Liukkonen E, Paetau R, Rekola M, Granström ML. Infantile spasms: diagnosis and assessment of treatment responseby video-EEG. Developmental medicine and child neurology. 2001 Oct;43(10):658-67.
15. Snyder HE, Jain P, RamachandranNair R, Jones KC, Whitney R. Genetic advancements in infantile epileptic spasms syndrome and opportunities for precision medicine. Genes. 2024 Feb 21;15(3):266.
16. Sumi MS, Sultana R, Khatun S, Chowdhury MS, Hussain ME, Chowdhury RN, Saha N. Clinical and Electroencephalographic Pattern and Short-Term Outcome to Treatment in Infantile Spasm: A Randomized Controlled Trial. Journal of National Institute of Neurosciences Bangladesh. 2022;8(2):130-5.
17. Menderes D, Serdaroğlu E, Hırfanoğlu T, Serdaroğlu A, Arhan E. What is the impact of etiology, lead time to treatment, and parental awareness on outcomes in infantile epileptic spasm syndrome?. Epilepsy & Behavior. 2025 Feb 1;163:110178.
18. Kumar Sahu J, Madaan P, Wanigasinghe J, Chand P, Winter S, Poudel P, Linn K, Fatema K, Min Aye AM, Samia P. Mitigating treatment lag for infantile epileptic spasms syndrome in low-and middle-income countries: Key recommendations from the South Asia allied IESS research group.
19. Vinters HV. Histopathology of brain tissue from patients with infantile spasms. International review of neurobiology. 2002 Jan 1;49:63-76.
20. Asadi-Pooya AA, Sharifzade M. West syndrome in South Iran: electro-clinical manifestations. Iranian journal of child neurology. 2013;7(3):40.
21. Bower BD, Jeavons PM. Infantile spasms and hypsarrhythmia. The Lancet. 1959 Mar 21;273(7073):605-9.
22. Fatema K, Akter N, Akhter S, Begum S, Rahman MH, Saad T. Pattern of EEG change in infants with West syndrome: a retrospective study. Epilepsy Journal. 2018;4(03).
23. HOEFER PF, deNAPOLI RA, LESSE S. Periodicity and hypsarrhythmia in the EEG: a study of infantile spasms, diffuse encephalopathies, and experimental lesions of the brain. Archives of Neurology. 1963 Oct 1;9(4):424-36.
24. Takatsu T. Japanese Society of Paediatric Neurology Nagoya City, May, 1965. Developmental Medicine & Child Neurology. 1965 Dec;7(6):705-8.
25. Hrachovy RA, Frost Jr JD, Kellaway P. Hypsarrhythmia: variations on the theme. Epilepsia. 1984 Jun;25(3):317-25.