ORIGINAL ARTICLE

Diversity in Clinical and Neurophysiological Manifestations of Epilepsy with Eyelid Myoclonia (Jeavons Syndrome) in Pediatric Population

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ABSTRACT

Objectives: Epilepsy with eyelid myoclonia (EEM), with or without absence, also known as Jeavons Syndrome (JS), is a unique epileptic syndrome. This syndrome may be accompanied by other generalized seizures, such as generalized, tonic-clonic, myoclonic, and rarely atomic seizures. This study was conducted to determine the diversity of clinical and neurophysiological manifestations of JS in the pediatric population of Iran.

Materials & Methods: This retrospective, cross-sectional study was conducted at the Children's Medical Center of Tehran, Iran, from 2017 to 2023. Two clinical neurophysiologists reviewed long-term video electroencephalographic (EEG) monitoring to confirm the diagnosis. Patients' demographic information was extracted from medical records or direct interviews based on clinical characteristics and history taking.

Results: Among 1530 patients admitted during the study period, 12 out of 17 previously diagnosed patients confirmed their diagnosis. Among the group of confirmed patients, seven were boys and five were girls. The average age of seizure onset was 3.4±1.7 years. Except for absence seizures, five out of 12 had no other types of seizures. Two patients showed generalized tonic-clonic events as associated seizures. One patient had astatic seizures, in the form of head drop, one patient had myoclonic seizures, and three patients had focal seizures without persistency in the EEG. Eleven of the 12 patients had focal electrographic findings during recording, with eight having focal epileptiform discharges during the interictal period.

Conclusion: JS is an under-recognized epileptic syndrome requiring accurate diagnosis through identifying seizure types and EEG features. Although it is classified as a generalized epilepsy, focal seizures have been reported in a few case reports and were observed in three patients during the ictal period in our study. Additionally, focal electrographic findings were prevalent during the interictal period. Further research is needed to better understand the clinical and neurophysiological aspects of this syndrome.

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Introduction

Several epileptic syndromes in idiopathic generalized epilepsy remain underrecognized or misdiagnosed. Epilepsy with eyelid myoclonia (EEM) is a distinctive and well-known disease within this group. Jeavons first described this syndrome in 1977

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(1). EEM characterizes itwith or without absence seizures, sensitivity to eye closure with epileptiform discharges or eyelid closure convulsions with epileptiform discharges, and sensitivity to light (photosensitivity) (2). EEM can be accompanied by absence seizures, known as EEMwith absence (ELMA), or it may be without absence seizures (ElM) (3). It is a unique epileptic disorder that typically presents in childhood, mostly between the ages of 6 and -8 years; however, earlier onset could be seen. In 2001, epilepsy with EEM (with/without absence) was first reported by the International League Against Epilepsy (ILAE) as a self-limiting seizure type with generalized onset (4). Later in 2010, it was recognized under generalized seizures as absence with specific characteristics (5). More recently, in 2017, it was recognized by the ILAE as a type of generalized non-motor seizure with eyelid myoclonus (6). The prevalence of Jeavons Syndrome (JS) is essential because this epilepsy constitutes 12.9% of generalized epilepsies, and its identification can improve clinical management and predict disease progression. Additionally, its diagnosis allows for the establishment of inclusion and exclusion criteria, leading to a clearer definition and better understanding of the true boundaries of other idiopathic generalized epilepsy syndromes (7,8,9). Even with effective seizure control, JS is a lifelong disorder. Approximately 20% of patients have a positive family history of epilepsy. The prevalence of the disease is higher in women, with a male-to-female ratio of approximately 1:2. The prognosis for men is generally better than for women. Neurological and cognitive development is typically normal prior to the onset of the disease (10,11).

Seizures in this syndrome are triggered by blinking and last for three to six seconds, occurring multiple times throughout the day (2). Intermittent light stimulation can induce seizures or photoconvulsive responses on EEG, although blinking is a stronger trigger than light stimulation (12, 13). Photosensitivity tends to decrease with age and treatment, unlike generalized tonic-clonic seizures, which usually occur with aggravating factors such as insomnia, drug and alcohol abuse, and persist throughout the disease course (14).

Generalized myoclonus is rare in JS. Seizures can be induced by voluntary or involuntary closing of the eyes or blinking as a reflex, but do not occur in darkness (2). Self-induction of seizures has been reported in some patients. While photosensitivity tends to disappear in middle age, EEM persists (15, 16, 17).

In some Western countries, JS is considered a long-standing disease, most commonly diagnosed in childhood (18, 19). Although in principle JS is not a focal epilepsy, EEG findings in JS have been described in some reports as focal occipital or frontal epileptiform discharges in both interictal and ictal phases (20, 21).

Focal seizures, originate in one hemisphere of the brain. They typically start with a single focus with excessive neuronal discharges that can spread to involve both hemispheres and become generalized seizures (22). Observing a focal seizure during video-EEG monitoring of a generalized epilepsy syndrome is not a usual finding.

Due to the limited global diagnosis of JS, there is a scarcity of available reports on the disease worldwide. Considering that most of the JS are not clearly identified, along with the difficulties in diagnosing and treating these patients, further contributes to the limited knowledge on this condition. Additionally, there have been no previous reports on the prevalence of JS in Iran. Therefore, this study aimed to investigate the demographic and neurophysiologic characteristics of JS, particularly focusing on focal neurophysiological findings, in a sample of pediatric population of Iranian patients.

Methods & Materials

This retrospective, cross-sectional descriptive study was conducted from 2017 to 2023 using 1530 long-term video-electroencephalographic monitoring (LTM) performed at the Epilepsy Monitoring Unit (EMU) of Children's Medical Center in Tehran, Iran.

All records were obtained using an EEG-Monitoring machine (NIHON KOHDEN, Neurofax, EEG-1200, Japan, 2014) and followed the standard 10-20 system for electrode placement, with additional T1 and T2 electrodes. Extra-leads, including surface electromyography (sEMG), electrocardiography (ECG), and right and left ocular leads, were also utilized. A low-frequency filter (LFF) of 1 Hz and a high-frequency filter (HFF) of 70 Hz were applied. To determine any focal abnormalities, all epochs were reviewed using different montages, including paired leads, paired group, and referential montages, specifically common average reference (CAR). In cases where focal abnormal findings were present, the epoch was re-read in referential montages to eliminate the effect of the phase cancellation phenomenon. During ictal events, both bipolar and monopolar montages were utilized, with special focus on the Pz electrode in patients who were asleep. At least two clinical neurophysiologists reviewed all records. In instances of disagreement, a consensus was reached through negotiation. Cases that could not reach a consensus were excluded from the study. The patients' demographic information was extracted from their medical records and through direct interviews in some

cases. The reviewed files included sleep-wake video and long-term EEG recordings, which were conducted for a minimum time of 24 hours to a maximum of 72 hours. The monitoring included sleep and wakefulness phases, as well as stimulation tests such as intermittent photic stimulation and hyperventilation (HV). Patients' demographic information was extracted from medical records or direct interviews based on clinical characteristics and history taking. This information includes the age of disease onset, family history of seizures, consanguinity of parents, developmental status before and after the onset of events, semiology of primary seizures, history of febrile seizures, and other useful demographic indices.

Results

Seventeen patients diagnosed with JS were identified in the EEG software information registry. These patients exhibited three common symptoms of JS, including eye closure sensitivity, photosensitivity, and EEM. However, after a thorough review of the VIDEO-EEGs and sleep-wake videos by two clinical neurophysiologists, only 12 patients were definitively confirmed to have their diagnosis.

Among the group of 12 confirmed patients, seven were boys and five were girls. Their ages ranged from 2.5 to 16 years old, with an average age of 7.3 ± 3.8 (median: 7) years. The onset of seizures in these patients occurred between 1.5 and 8 years old, with an average age of seizure onset at 3.4 ± 1.7 (median: 3) years (Table 1).

Regarding parental consanguinity, five patients had unrelated parents, three patients had cousins as parents, and four patients had distant relatives as parents. Three patients had a family history of epilepsy in first-degree relatives, while two patients had cousins with a history of epilepsy (Table 1).

Table 1. Demographic and clinical information of patients with JS

Semiology of primary seizures	Developmental status	History of febrile seizure	Consanguity Of parents	Family history of epilepsy	Age at onset of seizure	Gender	Age (years)	Patient
Generalized tonic	Abnormal	+	+	+	2	Male	11	1
Generalized tonic	Normal	-	-	-	4	Female	7	2
Focal tonic-clonic	Normal	+	-	+	2.5	Male	4.5	3
Eyelid myoclonia	Normal	-	+	+	2.5	Male	2 and 8 months	4
Generalized tonic-clonic	Abnormal	+	+	-	8	Female	16	5
Astatic(atonic) seizure	Abnormal	+	+	-	1.5	Male	2.5	6
Left hand focal tonic	Abnormal	-	-	-	7 months	Male	4 and 3 months	7
Eyelid myoclonia	Normal	-	-	+	3.5	Female	7 y and 8 months	8
Eyelid myoclonia	Normal	-	-	-	4	Male	6	9
Eyelid myoclonia	Normal	+	+	-	5	Female	9.5	10
Focal seizure	Abnormal	-	+	+	1.5	Female	9	11
Eyelid myoclonia	Abnormal	-	+	-	3	Male	7	12

Developmental status

In terms of developmental status, six patients exhibited normal development and achieved developmental milestones within the expected time. One patient showed mild developmental delay in the speech domain, while the remaining five patients showed significant developmental delay, particularly in speech development, socialization, and problem-solving. Approximately 50% of the patients showed developmental delay (Table 1).

Semiology of primary seizures

The semiology of primary seizures varied among the patients. Three patients experienced generalized tonic-clonic seizures, one patient had astatic seizures, and five patients presented with EEM. Additionally, three patients initially had focal seizures, which were later considered as associated seizures observed in long-term monitoring (Table 1). These focal seizures manifested as fencing position, lateral gaze with tonic phase, or focal clonic movements of the right hand, separate from EEM. In one patient, convulsions began before the age of two years, and metabolic tests did not reveal any specific metabolic disease. This patient experienced atonic convulsions starting at 1.5 years old, followed by EEM convulsions after the age of two years.

History of febrile seizure

Out of the 12 patients, five patients (3 boys and 2 girls) had a history of febrile seizures (Table 1). One patient experienced complex febrile convulsions that

were repeated several times, and focal seizures were observed along with JS. Another patient had simple febrile convulsions that started repeatedly with fever at the age of eight months and recurred every two to three months. In this patient, atonic seizures in the form of head drop started after 1.5 years of age, and EEM was also added at the age of two years.

Associated seizures

Except for absence epilepsy, a part of JS, which was seen in three patients, we observed generalized tonic-clonic, myoclonic, astatic, and focal seizures as associated seizures (Table 2). In contrast, focal seizures have not been widely reported as associated with JS in the literature; a few reports have been published about this association. Five out of 12 patients had no associated seizures. Two patients showed generalized tonic-clonic events as associated seizures. One patient had astatic seizures in the form of head drop, and one patient showed myoclonic seizures as jerky movements of the hands after EEM. Three patients showed focal seizures separately from EEM, including one patient with focal clonic seizures in the right hand and two patients with focal seizures manifested as fencing position and lateral gaze with tonic phase (Table 2).

Table 2. Neurophysiological findings of patients with JS

Patient	Age	Associated seizures	Sleep architecture	PDR (Posterior dominant rhythm)	Head Region of focal hints	Focal electrographic findings in EEG	Absence
1	11	None	disorganized	SLOWER THAN NORMAL	Anterior	RT and LT FT S&W	+
2	7	Generalized tonic-clonic	disorganized	Normal	Posterior	RT CTOS&W	+
3	4.5	Right sided focal clonic	Well organized	SLOWER THAN NORMAL	Posterior	Lt POS&W	-
4	2Y and 8 months	none	Well organized	Normal	Posterior + Anterior	RT and LT TO and FT	-
5	16	Generalized tonic-clonic	disorganized	Normal	Anterior	RT and LT	-
6	2.5	Astatic (atonic)epilepsy	Well organized	Normal	Anterior	RT F spike	-
7	4 y and 3	Focal tonic	Well organized	Normal	Anterior	RT FT and	-
8	7 y and 8 months	None	disorganized	SLOWER THAN NORMAL	Anterior	RT and LT FC S&W	-
9	6	None	Well organized	Normal	Posterior +	RT FT and	+
10	9.5	Myoclonic	Well organized	Normal	Anterior	RT FC	-
11	9	Focal seizure	Well organized	Normal	Posterior	Lt TO	-
12	7	None	Well organized	Normal	-	-	-

F=frontal C =central p=parietal, T=temporal, O=occipital, RT=right, LT = left and combination of these such as FT = Fronto-temporal, S&W= sharp or spike and wave

Focal electrographic findings during recording

In all patients, 3- to 6-Hz generalized multiple spike-and-wave discharges lasting almost 2-5 seconds, some with asymmetric features and associated eyelid myoclonus, were observed. Eleven out of 12 patients had focal electrographic findings during ictal or interictal recording, while one patient had no focal electrographic findings (Table 2). Eight out of 11 patients with focal findings had focal epileptiform discharges during the interictal period, and three out of 11 patients had focal electrographic findings during both the ictal and interictal periods. In the group with focal findings in the interictal period, two patients had focal epileptiform discharges only in the sleep state, three patients had them only in the awake state (one of them only with photic stimulation), and three patients had them in both the awake and sleep state.

Three cases presented with focal epilepsy as an associated seizure, and focal ictal activities were observed during focal seizures rather than EEM. In one case, in addition to generalized polyspike and wave discharges, focal epileptiform discharges during focal seizures were found to originate earlier in the temporal-occipital regions, with right temporal-occipital involvement in some events and left temporal involvement in the others (Figure 1, 2). This study classified focal abnormalities as anterior if localized in the frontal and fronto-central regions, and posterior if localized in the occipital, temporo-occipital, and parieto-occipital regions. Both interictal and ictal

(3/11) focal activity were observed. Six patients showed interictal focal activity in the anterior head region (Figure 3), three patients in the posterior region, and two patients were involved in both anterior and posterior head regions. One patient did not exhibit any focal EEG findings during recording (Table 2).

Sleep architecture

Wakefulness and sleep EEGs were performed in all patients. Sleep architecture was preserved in eight patients. However, in four patients, sleep EEGs were disorganized (Table 2).

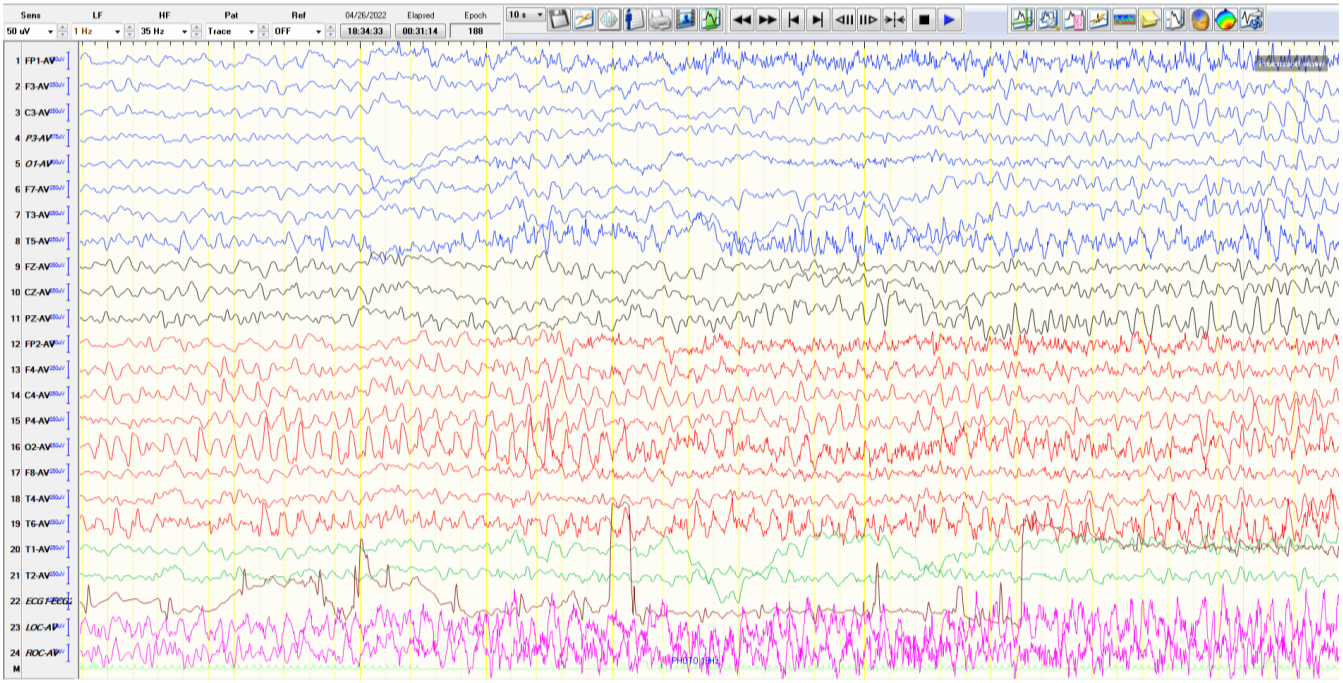

Figure 1. Referential montage (common average) shows focal ictal activity as evolution in right parieto-occipital region during a focal seizure in a nine years old girl with focal seizure and Jeavons syndrome

Discussion

EEM with or without absence seizures is often misdiagnosed or mistaken for tic or dream disorders (11). The primary reason for patients seeking medical attention is usually generalized tonic-clonic seizures, highlighting the underdiagnosis and lack of recognition of epilepsy with \EEM compared to its true prevalence.

In a previous study, patients with JS exhibited varying presentations of generalized tonic-clonic seizures throughout their lifetime. Some patients initially presented with generalized tonic-clonic seizures, while others did not experience any until after diagnosis of JS was made. Additional generalized seizure types, such as atonic, myoclonic, and tonic seizures, were also identified in some patients (21, 22).

Atonic and myoclonic seizures have been previously described in patients with JS, expanding on the clinical variability (21). Thus, a careful examination, particularly with video-EEG monitoring (VEM), is crucial for accurate diagnosis.

Although the predominance of females in JS has been well-documented, this study of 12 Iranian patients with JS out of 1530 long-term monitoring cases reported seven males (58.3%) and five females. This finding is not consistent with previous studies (11, 22, 23), such as one study conducted in the USA where 80% of 30 patients were female (10). The discrepancy may be attributed to differences in sample size and genetic factors among the patient populations. The present study found that the average age of seizure onset in patients was three years, which is consistent with previous studies.

While normal cognitive ability has been reported in individuals with JS, some may experience behavioral problems or have lower school achievement.

In this study, six patients had normal cognitive ability, while a study by Ashley et al. reported below-average cognitive abilities in all ten patients with JS, but mental retardation in these patients was not emphasized (24).

In the current study, seven people were of school age, of whom three (25%) reported success in school, and four (33 %) did not report success in school. Notably, more than half of the patients with JS in our report did not report success in school. At the same time, Joshi and Patrick reported a 77% school success rate in their patients (20).

Therefore, a more detailed assessment is needed to fully understand the cognitive aspects of this condition, which uncontrolled seizures and epileptic discharges may influence. Genetic factors have been discussed as potential contributors to the development of JS. Previous studies have reported a high prevalence (between 58% and 83%) of family history of epilepsy in patients with JS (11, 23), but in the present study, only 41% had a family history of epilepsy. This discrepancy suggests that further investigation is needed to determine the genetic basis of this syndrome and whether it is a separate epilepsy syndrome or not.

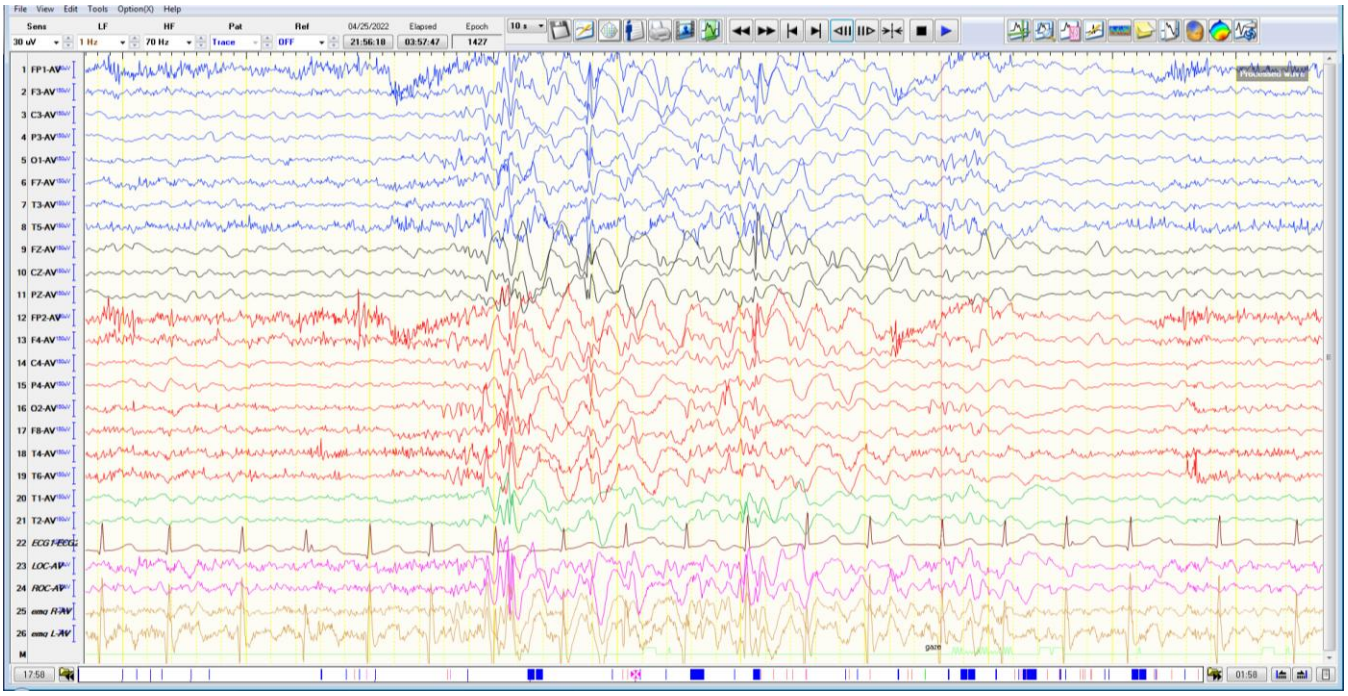

Figure 2. Referential montage (common average) shows diffuse polyspike and wave discharges following eye closure in favor of JS in a nine years old girl with focal seizure and JS.

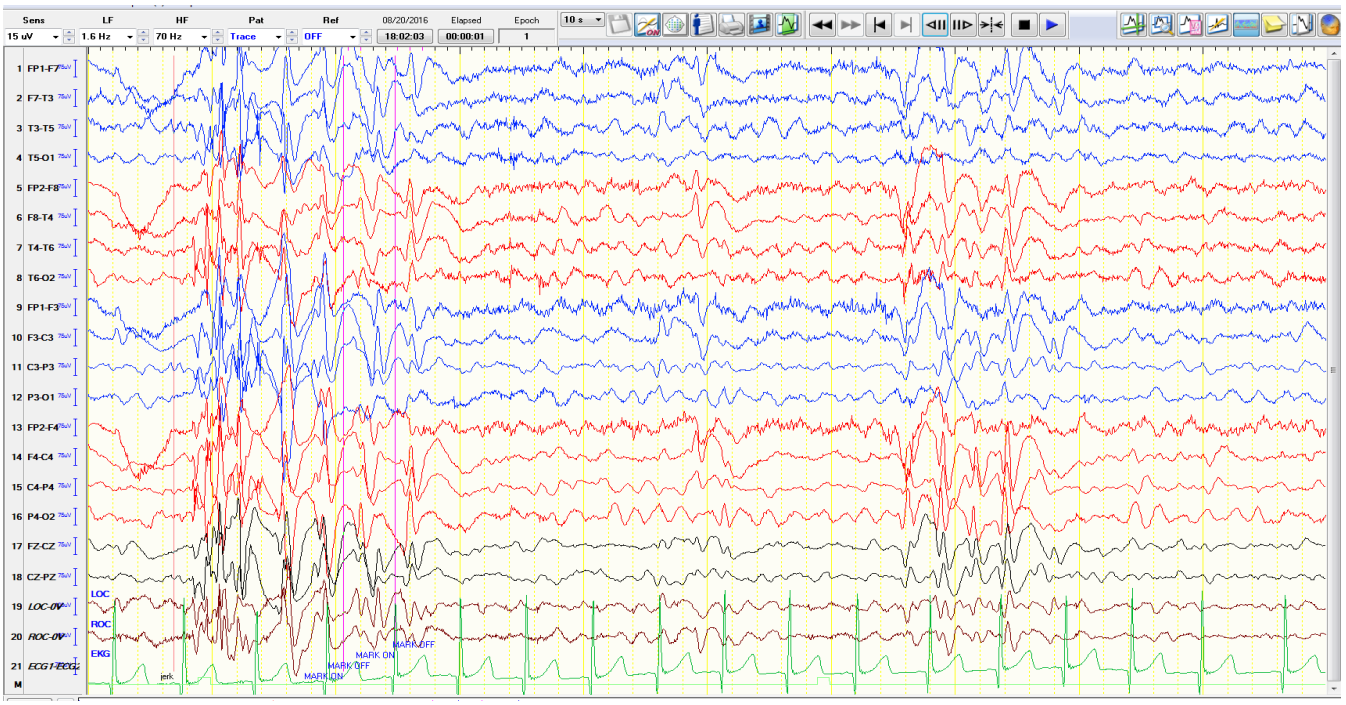

Figure 3. Anterior Posterior bipolar montage shows a focal interictal epileptiform discharge in right fronto-temporal region in an 11 years old boy with JS

Although JS is a well-defined epilepsy syndrome with clinical and EEG findings, it is considered by many authors to be an idiopathic generalized epilepsy, and its place in the ILAE classification is still unclear.

Panayiotopoulos suggested that it is a generalized idiopathic reflex epilepsy (2). Additionally, eyelid myoclonus with or without absence seizures can occur not only in JS but also in various types of epilepsy, such as idiopathic, symptomatic, or cryptogenic epilepsy (21).

Focal EEG findings are common in JS, which can potentially lead to misdiagnosis. In this study, out of 12 patients, 11 patients had focal EEG findings.

In another study, more than half of patients had focal EEG findings (22).

One study suggested that generalized epilepsy in JS originates from the occipital cortex due to posterior focal abnormalities detected on EEG (25). Senol et al. also found focal EEG findings in the occipital region in three of 31 patients with epilepsy with EEM and in the frontal region in four patients (26).

In this study, six patients showed the anterior head region, three the posterior head region, and two showed both the anterior and posterior head regions as the focal site of the electrographic region. This study could not define any persistent asymmetry or asynchrony during LTM patients' interpretation. Although JS is a reflex epilepsy and a type of generalized seizure, these findings are in favor of the prevalence of focal electrographic findings in JS, and further research is needed to better understand the significance of these focal EEG findings.

Notably, seizures and EEG abnormalities in JS are typically observed in the post-awakening period. In this study, sleep architecture was preserved in most patients, but irregular sleep patterns were observed in some, particularly those with associated seizures (Table 2). Additionally, it was observed that some important findings might be missed if EEG examinations are not performed after waking up. Another study also reported the presence of seizures and/or epileptiform discharges in post-awakening EEGs, even when pre-sleep and sleep-waking EEGs were normal (22). This highlights the importance of conducting EEG examinations after waking up to capture potential abnormalities.

In Conclusion

It is imperative to accurately diagnose epilepsy with EEM by distinguishing between seizure types and EEG features. However, further extensive prospective studies are necessary to validate and elucidate the demographic characteristics, unique clinical manifestations, and EEG findings associated with JS. Although JS is classified as a generalized epilepsy, focal seizures were observed during recording in three patients during the ictal period. Additionally, focal electrographic findings were prevalent during the interictal period. Hence, further research is needed to better understand the clinical and neurophysiological aspects of JS.

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Authors' Contribution

Study design and definite diagnosis of the cases: Hosein Eslamiyeh, Mahmoud Mohammadi, Reza Shervin Badv, Meharn Beiraghi Toosi. Interpretation of data and revision of the manuscript: Gholam Reza Zamani , Morteza Heidari1, Zahra Rezaei .

Conflict of Interest

The author declared no conflict of interest

References

1. Jeavons PM. Nosological problems of myoclonic epilepsies in childhood and adolescence. Dev Med Child Neurol 1977;19(1):3–8.
2. Panayiotopoulos CP. Syndromes of idiopathic generalized epilepsies not recognized by the International League Against Epilepsy. Epilepsia 2005;46 Suppl 9:57–66.
3. Giráldez BG, Serratosa JM2. Jeavons syndrome as an occipital cortex initiated generalized epilepsy: Further evidence from a patient with a photic-induced occipital seizure. Seizure 2015;32:72–4.
4. Engel J Jr; International League Against Epilepsy (ILAE). A proposed diagnostic scheme for people with epileptic seizures and with epilepsy: report of the ILAE task force on classification and terminology. Epilepsia. 2001;42(6):796–803. doi:10.1046/j.1528-1157.2001.10401.x
5. Berg AT, Berkovic SF, Brodie MJ, Buchhalter J, Cross JH, van Emde Boas W, Engel J, French J, Glauser TA, Mathern GW, et al. Revised terminology and concepts for organization of seizures and epilepsies: report of the ILAE commission on classification and terminology, 2005-2009. Epilepsia. 2010;51(4):676–685.
6. Fisher RS, Cross JH, French JA, Higurashi N, Hirsch E, Jansen FE, Lagae L, Moshé SL, Peltola J, Roulet Perez E, et al. Operational classification of seizure types by the international league against epilepsy: position paper of the ILAE commission for classification and terminology. Epilepsia. 2017; 58(4):522–530.
7. Covanis A. Eyelid myoclonia and absence. Adv Neurol. 2005;95:185–196.
8. Covanis A. Jeavons syndrome – updated review. J Epileptology. 2015;23(2):113–123.
9. Smith KM, Youssef PE, Wirrell EC, Nickels KC, Payne ET, Britton JW, Shin C, Cascino GD, Patterson MC, Wong-Kisiel

LC. Jeavons syndrome: clinical features and response to treatment. Pediatr Neurol. 2018;86:46–51.
10. Smith KM, Youssef PE, Wirrell EC, Nickels KC, Payne ET, Britton JW, Shin C, Cascino GD, Patterson MC, Wong-Kisiel LC. Jeavons syndrome: clinical features and response to treatment. Pediatr Neurol. 2018;86:46–51.
11. Wang XL, Bao JX, Liang-Shi T-M, Deng YC, Zhao G, Swa B, Liu YH. Jeavons syndrome in China. Epilepsy Behavior. 2014;32:64–71.
12. Giannakodimos S, Panayiotopoulos CP. Eyelid myoclonia with absences in adults: a clinical and video-EEG study. Epilepsia 1996;37(1):36–44.
13. Striano S, Striano P, Nocerino C, Boccella P, Bilo L, Meo R, et al. Eyelid myoclonia with absences: an overlooked epileptic syndrome? Neurophysiol Clin 2002;32(5):287–96.
14. .Striano S, Capovilla G, Sofia V, Romeo A, Rubboli G, Striano P, et al. Eyelid myoclonia with absences (Jeavons syndrome): a welldefined idiopathic generalized epilepsy syndrome or a spectrum of photosensitive conditions? Epilepsia 2009;50 Suppl 5:15–9.
15. .Pérez-Errazquin F, Chamorro-Muñoz MI, García-Martín G, Romero-Acebal M. Does Jeavons syndrome exist? A report of a series of 10 cases. Rev Neurol 2010;50(10):584–90.
16. Viravan S, Go C, Ochi A, Akiyama T, Carter Snead III O, Otsubo H. Jeavons syndrome existing as occipital cortex initiating generalized epilepsy. Epilepsia 2011;52:1273–9.
17. Striano S, Capovilla G, Sofia V, Romeo A, Rubboli G, Striano P, et al. Eyelid myoclonia with absences (Jeavons syndrome): a well-defined idiopathic generalized epilepsy syndrome or a spectrum of photosensitive conditions. Epilepsia 2009;50(Suppl.5):15–9.
18. Fernandes MJS. Epilepsia do lobo temporal: mecanismos e perspectivas. Estud Av. 2013;27(77):85-96. doi: 10.1590/S0103-40142013000100007.
19. Abou-Khalil, B.W., Antiepileptic Drugs. Continuum (Minneap Minn), 2016. 22(1 Epilepsy): p.132-56.
20. Joshi, C.N. and J. Patrick, Eyelid myoclonia with absences: routine EEG is sufficient to make a diagnosis. Seizure, 2007. 16(3): p. 254-60.
21. Dragoumi, P., et al., Crossing the lines between epilepsy syndromes: a myoclonic epilepsy variant with prominent eyelid myoclonia and atonic components. Epileptic Disord, 2018. 20(1): p. 35-41.
22. Selma TOPALOĞLU TUAÇ, Cengiz YALÇINKAYA, Ahmet Veysi DEMİRBİLEK. Jeavons Syndrome: 12 Cases. Epilepsia 2016;22(2):72-76
23. Sadleir, L.G., et al., Family studies of individuals with eyelid myoclonia with absences. Epilepsia, 2012. 53(12): p. 2141-8.
24. Fournier-Goodnight AS, Gabriel M, Perry MS. Preliminary neurocognitive outcomes in Jeavons syndrome. Epilepsy Behav 2015;52(Pt A):260–3.
25. Viravan S, Go C, Ochi A, Akiyama T, Carter Snead O 3rd, Otsubo H. Jeavons syndrome existing as occipital cortex initiating generalized epilepsy. Epilepsia 2011;52(7):1273–9.
26. Nar Senol P, Tezer FI, Saygi S. Eyelid myoclonia seizures in adults: An alternate look at the syndrome paradox. Epilepsy Behav 2015;45:265–70.