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Evaluating the Effects of Medication on the ESES Patterns and Autism Traits in Non-Epileptic Children with ASD in Bandar Abbas, Iran

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Abstract

Background: Autism is a disorder that affects brain development, causing difficulties in social interaction, communication, and interests. ESES is a sleep-related condition linked to autism. This study examined the epileptic conditions in autistic children, their clinical response, and brain wave normalization following medication treatments.

Methods: This clinical trial was conducted on children with autism who had initial EEG abnormalities during sleep. The children were divided into three distinct groups, each administered different medications. A pediatric neurologist monitored the children's autistic symptoms using ESES assessments over 6 months. Ultimately, the effect of different medications on improving the children's autistic symptoms and EEG patterns was evaluated.

Results: Out of the 29 patients who participated in this study, 6 (20.7%) were completely removed from the autism spectrum, 21 (72.4%) showed a clinical response of more than 50%, and 2 (6.9%) did not show any clinical response. Additionally, the group administered sodium valproate showed the greatest improvement compared to the other groups. Regarding the final EEG results, 3 cases (10.3%) showed complete normalization, 24 cases (82.8%) exhibited suitable changes, and 2 cases (6.9%) remained unchanged. However, these changes were not statistically significant.

Conclusion: This study demonstrated that sodium valproate and corticosteroids can improve EEG alterations in ESES and, as a result, reduce children's autistic symptoms. However, due to the lack of statistical significance in the data, it is suggested that additional trials with larger sample sizes and longer follow-up periods be conducted to obtain more reliable results.

Keywords: Autism spectrum disorders, Electroencephalography, Status epilepticus, Pediatric

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Introduction

Autism spectrum disorder (ASD) is a chronic neurodevelopmental syndrome characterized by difficulties in social communication and language, as well as the presence of restricted and repetitive activities (1,2). ASD is categorized as a spectrum disorder because of its diverse expressions among patients. These anomalies typically emerge early in life and often result in functional deficits in various environments (2). The Centers for Disease Control estimates that 1 in 59 children, regardless of racial, cultural, or socioeconomic background, have been diagnosed with ASD (3).

Current research suggests that most children with ASD might be diagnosed before 24 months of age (4-6). There has been extensive research into the possible causes of autism using pharmacological, pathological, electrophysiological, and imaging methods, but the underlying cause remains unknown. The presence of several neurological disorders associated with autism, significant changes in the nervous system, and expanding research on structural and functional differences in the brains of people with autism suggest a biological dysfunction in the central nervous system (CNS). Additionally, there is an association between CNS malfunctions and increased susceptibility to seizures (7). Furthermore, research has shown that specific autoimmune and paraneoplastic disorders may have similarities to autism spectrum disorders. These syndromes include Electrical Status Epilepticus in Sleep (ESES), Landau-Kleffner Syndrome (LKS), and Continuous Spike and Wave during Slow Wave Sleep (CSWS). Additionally, when these children undergo an EEG, both LKS and CSWS may show ESES patterns

(8). According to reports, one-third of people with ASD experience this condition (7). Nevertheless, the precise occurrence of this phenomenon is still uncertain, and the available research provides a broad spectrum of estimates, ranging from 5% to 46% (9,10). Autism is not accompanied by a specific type or spectrum of seizures. There have been reports of absence seizures, generalized tonic-clonic seizures, and complex partial seizures (with or without secondary generalization) (11-15). Diagnosing seizure activity in autistic individuals is challenging because behavioral abnormalities linked to complex partial and/or absence seizures, such as staring and non-responsiveness with or without repetitive motor activities, can be attributed to autism. Current studies indicate a significant occurrence of epileptiform electroencephalograms (EEGs) in children with autism who do not have a prior medical history of seizures or epilepsy (16,17). Tuchman et al demonstrated that children with significant intellectual disabilities exhibit a higher prevalence of seizures. Children with autism are 30% more vulnerable to epilepsy, which is generally associated with cognitive impairment (18). Although the necessity of immediately handling convulsive status epilepticus has been definitively proven (19), it remains uncertain whether the treatment of sleep-induced status epilepticus, a neurophysiological characteristic of ESES syndrome, will delay long-term cognitive deterioration. Generally, decisions for treatment often follow the recommendations of experts. Studies on the effects of antiepileptic medications, benzodiazepines, steroids, intravenous immunoglobulins, the ketogenic diet, and epilepsy surgeries on electroencephalograms (EEGs) and cognitive functioning have been suggested (20).

This study aimed to investigate the prevalence of epileptic disorders in autistic children and the extent of clinical response and EEG normalization following pharmacological interventions.

Methods

This quasi-experimental study was conducted to investigate the effect of treatment in autistic children without a history of epilepsy on primary EEG disorders and autistic symptoms. To conduct this study, we analyzed every child aged 1 to 15 years with ASD in a non-random manner who had been referred to the pediatric neurology clinic or the neurology clinic of Bandar Abbas Children's Hospital in 2018.

Study design and sampling

To begin, the samples were chosen in a non-random manner according to the study's inclusion and exclusion criteria. The EEGs were taken from the patients while they were in deep sleep (non-rapid eye-movement (NREM) sleep) (21).

We initially examined 69 known cases of autism. The study excluded all 23 cases with a history of clinical seizures. We examined the remaining cases without a history of clinical seizures using EEGs. Of these, 17 cases were excluded because their EEGs were completely normal. We monitored the remaining 29 cases for 6 months for EEG abnormalities and prescribed appropriate medications. Two pediatric neurologists, who were unaware of the study process, reported and confirmed all cases with EEG abnormalities (22). As the EEG interpretation criteria require brain waves to have normal amplitude and frequency, the neurologists interpreted the cases if they observed deviations from these normal values.

The remaining patients were divided into three groups based on the type of disorder in the EEG:

Group A (n = 11): ASD patients with non-specific epileptiform waves, memory disorder, and learning disorder in the EEG during sleep, group B (n = 11): ASD patients with high voltage slow wave (HVSW) or LKS observed during sleep, group C (n = 7): ASD patients with CSWS waves observed in the EEG during sleep.

The patients in the three groups were treated as follows

- Group A: Treated with sodium valproate at a rate of 20 mg/kg.
- Group B: Treated with pulse methylprednisolone (15-33 mg/kg) for 3 days, followed by continuous treatment with oral corticosteroid at a rate of 1-2 mg/kg/day.
- Group C: Treated with oral corticosteroid at a rate of 1-2 mg/kg/day.(23).

In the next stage, EEGs were taken from the patients monthly for 6 months. The EEG interpretation and improvement of clinical symptoms were performed by pediatric neurologists. The final EEG changes were classified as completely normalized, partially normalized, or unchanged. Clinical results were reported as a complete departure from the autism spectrum, more than 50% improvement in symptoms, or no change.

Inclusion and exclusion criteria

Inclusion criteria comprised all children aged between 1 and 15 years with autism spectrum disorder who were referred to the Pediatric Neurology Clinic or the Neurology Clinic at Bandar Abbas Children's Hospital during the second half of 2018. Exclusion criteria included patients who had not experienced seizures before the start of the procedure and patients whose initial brain scans were normal. It should be mentioned that all eligible patients who met the inclusion and exclusion criteria and were referred to the pediatric neurology clinic of Bandar Abbas Children's Hospital during the study period were included. Considering the rarity of ASD patients without a history of seizures but with abnormal EEG findings, it was not feasible to recruit a larger cohort within the available timeframe.

Data analysis

In this study, statistical tests (Kruskal-Wallis and Kolmogorov-Smirnov for nonparametric variables, and Chi-square for nominal variables) were used to evaluate the recorded results. A P value of ≤ 0.05 was considered significant. The data were analyzed using SPSS version 22.

In this study, statistical analyses were performed using SPSS version 22. For continuous variables, the Kruskal-Wallis test was applied to compare the three groups, while the Kolmogorov-Smirnov test was used to assess normality. For categorical variables, Fisher's exact test was employed instead of the Chi-square test due to the small sample size and expected frequencies in some categories. A P value ≤ 0.05 was considered statistically significant.

Ethical considerations

This investigation was derived from a thesis submitted to achieve a general doctorate in medicine. The study and data collection from patients referred to the Children's Neurology Service at Bandar Abbas, which is affiliated with Hormozgan University of Medical Sciences and Medical Services, and received the Ethical code: IR.HUMS.REC.1398.349, was carried out in 2018 with approval from the Ethics Committee of Hormozgan University of Medical Sciences and Health Services, Bandar Abbas. Following a thorough explanation of the study's methods, the parents of the patients gave their signed informed consent after being assured of the study's confidentiality.

Results

The study included a population of 29 patients diagnosed with ASD, of whom 17 were male. Table 1 displays data on the gender and distribution of primary developmental disorders among the participants.

The study included patients aged between 1 and 15 years. The results indicated that the average age of the entire population was 4.34 ± 2.88 years (Median = 3, Interquartile range: 2-6). Among the male participants, the average age was 4.17 ± 1.91 years (Median = 4, Interquartile range: 2.5-5.5), while among the female participants, the average age was 4.58 ± 3.96 years (Median = 3, Interquartile range: 2-6). There was no significant difference between the two sexes in terms of age (Z = -0.65; P = 0.521). The mean age was 4.54 ± 2.25 (Median = 4, Interquartile range: 3-7), 4.54 ± 3.8 (Median = 3, Interquartile range: 2-6), and 3.71 ± 2.36 (Median = 3, Interquartile range: 2-6) in groups A, B, and C, respectively. There was no statistically significant difference in mean age between the three groups (Z = -1.16; P = 0.560).

In general, 13 patients (44.82%) had Speech-Cognitive-Behavioral Disorder, while 16 patients (55.17%) had Speech-Cognitive Disorder. The other results of the present study showed no significant difference between the three groups in terms of Speech Cognitive Behavioral Disorder (χ^2 = 1.16, P = 0.560) and Speech-Cognitive Disorder (χ^2 = 1.16, P = 0.560).

Table 1. Demographic information of patients

Variable	Groups	Class	No. (%)	Statistical analysis*	*P* value
Sex	Group A	Male	7(63.6%)	0.94	0.708
		Female	4(36.4%)		
	Group B	Male	7(63.6%)		
		Female	4(36.4%)		
	Group C	Male	3(42.9%)		
		Female	4(36.4%)		
Speech-Cognitive-Behavioral disorder	Group A	Yes	6 (54.5%)	2.22	0.443
		No	5 (45.5%)		
	Group B	Yes	3 (27.3%)		
		No	8 (72.8%)		
	Group C	Yes	4 (57.1%)		
		No	3 (42.9%)		
Speech-cognitive disorder	Group A	Yes	5 (45.5%)	2.22	0.443
		No	6 (54.5%)		
	Group B	Yes	8 (72.7%)		
		No	3 (27.3%)		
	Group C	Yes	3 (42.9%)		
		No	4 (57.1%)		

* Exact Fisher test

The findings indicated that there was no statistically significant clinical response among the patients (P = 0.374), as shown in Table 2. In total, according to the results obtained from our study, 6 patients (20.7%) were completely removed from the autism spectrum, 21 (72.4%) children showed more than a 50% clinical response, and finally, 2 (6.9%) patients had no clinical response. Figure 1 presents the clinical response across different groups.

In the final recorded EEG, the results showed that among the patients, 3 cases (10.3%) were completely normal, 24 patients (82.8%) had no initial changes, and 2 patients (6.9%) were unchanged. Table 3 displays the impact of the prescribed medications on the final EEG for each of the groups. The results indicated that there was no statistically significant relationship between the final EEG results and the clinical response (P = 0.876). While sodium valproate and corticosteroids administered individually could enhance the EEG changes in ESES and therefore alleviate autistic symptoms in patients, these effects did not reach statistical significance. Figure 2 shows the final EEG results across different groups.

Discussion

Our general aim in conducting this study was to examine children with autism referred to medical centers to identify those with epilepsy-like disorders (ESES) by recording their brain waves during sleep. Additionally, to reduce or eliminate these disorders in the EEG, we

Table 2. Clinical response in each group

Variable	Group A (n=11)	Group B (n=11)	Group C (n=7)	Total (n=29)	Statistical analysis*	*P* value
Departure from the ASD spectrum	4 (36.4%)	1 (9.1%)	1 (14.3%)	6 (20.7%)		
>50%	6 (54.5%)	9 (81.8%)	6 (85.7%)	21 (72.4%)	3.58	0.572
Without a clinical response	1 (9.1%)	1 (9.1%)	0 (0.0%)	2 (6.9%)		

* Exact Fisher test.

Table 3. The final EEG results in each group

Variable	Group A (n=11)	Group B (n=11)	Group C (n=7)	Total (n=29)	Statistical analysis*	*P* value
Normal	2 (18.2%)	1 (9.1%)	0 (0.0%)	3 (10.3%)		
No initial change	8 (72.8 %)	9 (81.8%)	7 (100%)	24 (82.8%)	2.41	0.843
Without change	1(9.1%)	1(9.1%)	0(0.0%)	2(6.9%)		

* Exact Fisher test.

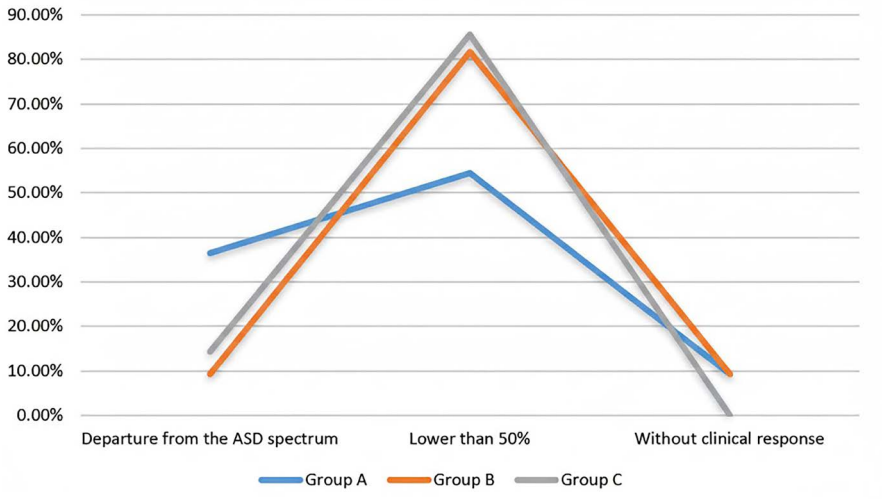

Figure 1. The frequency of clinical response in each group

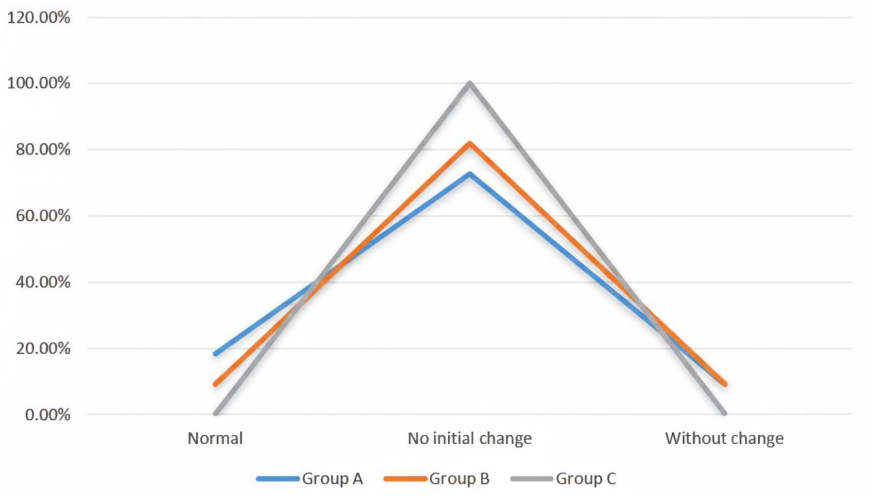

Figure 2. The final EEG results in each group

investigated the effectiveness of using sodium valproate and corticosteroid drugs in improving EEG abnormalities and facilitating the departure from the autism spectrum.

Although the findings of our study were not statistically significant, we observed changes in the perspective of the effects of corticosteroids. The small size of the studied population accounts for this lack of significance. Some studies have reported disease recurrence at varying

intervals after corticosteroid use, which contradicts our study's findings. This discrepancy could be a significant factor, considering our study's length compared to shorter studies. Regarding the effect of sodium valproate, the findings of our study cannot be compared with those of other studies. The reason for this is that most of the studies were conducted retrospectively, and none of them investigated the effect of sodium valproate in patients alone. Instead, our study evaluated the effectiveness of several anticonvulsant drugs alone or in combination with other drugs beyond the anticonvulsant spectrum, yielding variable results. Another difference that may make it difficult to accurately compare the results of the present study with those of previous texts is the grouping of patients based on their initial brain scan findings and the selection of a specific drug for each of these groups.

In a retrospective study, Wiwattanadittakul et al investigated ESES, treatment patterns, and EEG outcomes. The results showed that initially, 76% of individuals with electrical status epilepticus in sleep experienced resolution, but 56% of them later experienced a relapse. The rate of relapse was much greater for steroids (89%) and benzodiazepines (60%) compared to nonbenzodiazepine antiepileptic drugs (29%) (24). Rousselle et al (25) distinguished three groups based on the neuropsychological status attained during the ESES phase. Children in the first group were neuropsychologically healthy, children in the second group were diagnosed with verbal auditory agnosia, and children in the final group were cognitively and neuropsychologically impaired overall. The predominant epileptogenic target among children with aphasic symptoms has been temporal, while it was frontal in those with general cognitive impairment. Nonetheless, it is widely recognized that either positive or negative myoclonus may be linked to frontal spikes (26). Therefore, certain clinical variances with behavioral or motor abnormalities may be identified based on the placement of the spikes. Furthermore, focal ESES could be the single EEG sign for the epileptic encephalopathy associated with ESES, as demonstrated by Carraballo et al. According to their observations, focal ESES may represent a recurrence of the ESES phase or be a component of the traditional ESES syndrome during the initial ESES period that develops into diffuse ESES (27). Some researchers believe that, as poor neuropsychological performance is the case with the classic ESES syndrome, it may be explained by faulty synapse formation as a result of dramatically altered neuronal activity during a key phase for synaptogenesis (28).

In a study investigating the use of amantadine for treating ESES, the results indicated that it was somewhat effective for ESES-related syndromes. Moreover, the post-amantadine spike-wave index (53%) showed a significant reduction compared to the median baseline spike-wave index (76%). Thirty percent of patients experienced either complete or near-complete treatment of ESES. The majority of patients demonstrated subjective improvements in cognitive, linguistic, or behavioral functioning (29).

The retrospective study on patients with typical clinical manifestations of the ESES syndrome who were treated with anticonvulsant drugs (single drugs or combinations of drugs) found that all single drug treatments, including sodium valproate, were not effective, and only lotiracetam had a transient response (27). Another study revealed that lacosamide is a reliable AED that could benefit more than half of CSWS patients with their electroclinical impairment (30).

Another study reported that children with CSWS who are refractory to other typical AEDs and have symptomatic epilepsy, whose paroxysmal action has a regional location without secondary bilateral synchrony (SBS), may benefit from levetiracetam as an add-on therapy at a dosage of 45–50 mg/kg/day (31).

It could be mentioned that continuous paroxysmal activity in a specific area of the brain during sleep may lead to cognitive dysfunction. This discovery suggests that it has similarities to bilateral or diffuse electrical status epilepticus in sleep (ESES) in terms of causing neurological decline (25,26,28,32). Therefore, the epileptiform discharges that occur during a specific stage of sleep, independent of how they are spread out, coordinated or uncoordinated, and their structure, may have a significant impact on the decline of cognitive function (28, 33-37). It would be noteworthy to examine the role of this atypical EEG activity during this stage of sleep in the progression of deterioration, as slow waves were also identified in several patients in the study with bilateral brain involvement (38, 39).

It is worth mentioning that one of the most important factors to consider, according to the results of our study, is that ESES requires rapid diagnosis and aggressive treatment. Additionally, regular follow-ups and serial brain scans during sleep are necessary to monitor the response to treatment.

These findings provide initial insights into EEG-related abnormalities in ASD patients. However, some limitations should be acknowledged. First, prolonged follow-up is essential for identifying the recurrence of EEG-related abnormalities and associated clinical symptoms. Therefore, conducting clinical trials with extended observation periods is recommended. Moreover, evaluating these abnormalities through the calculation of the spike-wave index can provide more precise insights into EEG disorders by allowing comparison between baseline findings and post-treatment changes, thereby offering researchers a broader perspective. Another notable limitation of the present study is the relatively small sample size (n = 29). This was primarily due to the strict inclusion criteria, which only permitted the

enrollment of ASD patients without prior seizures but with abnormal EEG results. Such restrictions inevitably limited the statistical power of the analyses and may account for the absence of statistically significant results despite observable clinical improvements. Future studies with larger, multi-center populations are needed to confirm and strengthen these preliminary observations.

Conclusion

In conclusion, this study demonstrated that the administration of sodium valproate and corticosteroids as a single medication can enhance EEG alterations in ESES and subsequently alleviate autism symptoms in patients. However, it is important to note that these alterations did not reach statistical significance. Furthermore, our findings indicated a lack of correlation between age and the clinical response observed in patients diagnosed with autism.

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Supervision: Hashem Lashgari.
Validation: Hashem Lashgari.
Visualization: Fateme Fazli.
Writing–original draft: Raziye Najafabadi Pour.

Competing Interests

The authors declare no conflict of interest.

Ethical Approval

The study was approved by the Ethics Committee of Hormozgan University of Medical Sciences and Health Services, Bandar Abbas (Ethical Code: IR.HUMS.REC.1398.349) in 2018.

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