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SEIZURE SPECTRUM IN CHILDREN WITH SUBACUTE SCLEROSING PANENCEPHALITIS: A CROSS-SECTIONAL STUDY FROM A TERTIARY CARE CENTER

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ABSTRACT

Background and Objective:

Subacute sclerosing panencephalitis (SSPE) is a progressive and fatal neurodegenerative disorder caused by persistent infection of the central nervous system with a mutated measles virus. The objective of this study was to describe the spectrum and electroclinical characteristics of seizure types in children with SSPE.

Methods:

This retrospective observational study was conducted at a tertiary-care pediatric neurology center in Pakistan from January 2020 to December 2025. Children aged 2–14 years fulfilling modified Dyken's criteria for SSPE were included. Clinical records, electroencephalography (EEG), and laboratory data were reviewed. Seizure semiology and electroclinical features were analyzed, and seizures were classified according to the International League Against Epilepsy 2017 criteria.

Results:

A total of 125 children were analyzed (male: female ratio 3:1; mean age 7.1 ± 2.8 years). Myoclonic seizures were most frequent (52.8%), followed by movement disorder–associated paroxysmal events in the form of dystonia (21.6%), generalized tonic–clonic seizures (13.6%), atonic seizures (5.6%), and focal seizures (4.0%). EEG showed no focal or lateralized epileptiform activity; rather, all patients exhibited generalized periodic high-amplitude slow-wave complexes, frequently time-locked with myoclonic jerks. Seizures were frequent and pharmacoresistant, with sustained seizure control achieved in only 6.4% of patients. Severe postictal impairment was observed in 65.6% of cases.

Conclusion:

Children with SSPE exhibit a distinctive epileptic phenotype dominated by myoclonic and dystonic posturing with characteristic EEG correlates. Early recognition of these patterns aids diagnosis, while effective measles immunization remains the only proven preventive strategy.

Keywords:

Electroencephalography; Myoclonic seizures; Seizure spectrum

INTRODUCTION

Subacute sclerosing panencephalitis (SSPE) is a progressive and fatal neurodegenerative disorder caused by persistent infection of the central nervous system with a mutated measles virus.[1] The disease typically manifests several years after primary measles infection and reflects a failure of viral clearance from neuronal and glial cells.[2] Ongoing viral persistence leads to chronic neuroinflammation, demyelination, and progressive neuronal loss, resulting in widespread cortical and subcortical dysfunction. Despite being preventable, SSPE continues to be encountered in pediatric neurology practice in Pakistan, reflecting ongoing gaps in measles control.[3]

The incidence of SSPE has declined substantially in countries with sustained high measles vaccination coverage; however, it remains a significant neurological problem in low- and middle-income regions.[4] Pakistan continues to report measles outbreaks due to incomplete immunization, delayed vaccination, and health system challenges, which collectively contribute to a continued

burden of SSPE.[5] In routine clinical practice, many affected children have either missed vaccination or have undocumented immunization status, underscoring persistent gaps in preventive healthcare delivery.

Epidemiological studies have consistently demonstrated that measles infection occurring before two years of age confers the highest risk for subsequent development of SSPE.[6] Consequently, SSPE remains a frequently encountered entity in pediatric neurology practice in Pakistan, often presenting at advanced stages with significant neurological morbidity.

Clinically, SSPE follows a well-recognized staged progression, classically described by Jabbour et al.[7] Stage I is characterized by subtle behavioral changes, cognitive decline, and academic deterioration, which are often mis-attributed to psychosocial or educational issues. Stage II marks the emergence of myoclonic jerks, seizures, and progressive motor dysfunction, frequently prompting neurological evaluation. Stage III is dominated by severe extrapyramidal manifestations, rigidity, dystonia, and loss of ambulation, while Stage IV culminates in a vegetative or decerebrate state leading to death.[8] Among these stages, seizures, particularly myoclonic seizures, are most prominent during Stages II and III and often dominate the clinical course.

Seizures represent one of the most disabling manifestations of SSPE and frequently prompt referral to pediatric neurology services.[9] Although myoclonic jerks are widely regarded as characteristic, the seizure phenotype in SSPE is often heterogeneous and may evolve as the disease progresses.[10] Generalized tonic–clonic, atonic, focal seizures, and abnormal hyperkinetic events may coexist, particularly in advanced stages.[11]

Electroencephalography (EEG) remains a cornerstone of diagnosis, with periodic, bilaterally synchronous slow-wave complexes providing a strong electroclinical correlation, especially when time-locked with myoclonic activity.[12] These periodic discharges frequently correlate temporally with myoclonic jerks and evolve with disease progression, offering both diagnostic and prognostic value.[13] Early misinterpretation of seizure semiology may result in diagnostic delay or misclassification as other epileptic encephalopathies, especially in resource-limited settings.

Given the continued occurrence of SSPE in Pakistan and the limited local data describing its epileptic manifestations, this study aimed to characterize the spectrum of seizure types and associated electroencephalographic features in children diagnosed with SSPE at a tertiary-care pediatric neurology center.

METHODS

This retrospective cross-sectional observational study was conducted at the Department of Paediatric Neurology, The Children's Hospital & Institute of Child Health, Multan, from January 2020 to December 2025. Ethical approval was obtained from the Institutional Ethical Review Committee (no. 1963, dated 1st October 2025), and the study was conducted in accordance with the Declaration of Helsinki.

Children aged 2–14 years diagnosed with SSPE according to modified Dyken's criteria were included using consecutive sampling. The diagnostic criteria comprised progressive neurological deterioration, characteristic electroencephalographic findings, and elevated anti-measles antibody titers in cerebrospinal fluid. But due to non-availability, this test was not performed in our cohort. Each EEG recording was performed for a minimum duration of 20–30 minutes. Patients with alternative neurological diagnoses were excluded.

Demographic characteristics, vaccination status, clinical features, seizure semiology, electroencephalographic findings, and response to antiseizure medications were extracted from standardized medical records. Seizures were classified according to the International League Against Epilepsy (ILAE) 2017 operational classification. Electroencephalography was performed in all patients using the international 10–20 system and interpreted by consultant pediatric neurologists.

Data were analyzed using SPSS version 26.0. Continuous

variables were expressed as mean ± standard deviation, while categorical variables were summarized as frequencies and percentages. Associations between categorical variables were explored using the Chi-square test or Fisher's exact test, as appropriate. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 125 children aged 2–14 years with subacute sclerosing panencephalitis were included in the analysis. There was a marked male predominance, with 94 males (75.2%) and 31 females (24.8%), yielding a male-to-female ratio of approximately 3:1. The mean age at presentation was 7.1 ± 2.8 years. Most patients belonged to low socioeconomic backgrounds, and a substantial proportion had incomplete or undocumented measles vaccination history. Baseline demographic and clinical characteristics are summarized in Table 1.

Table 1. Demographic and Clinical Characteristics of Children with SSPE (n = 125)

Variable	Value
Age (years), mean ± SD	7.1 ± 2.8
Age range (years)	2–14
Male sex	94 (75.2%)
Female sex	31 (24.8%)
Seizures present	121 (96.8%)
Seizures as presenting symptom	72 (57.6%)
Severe postictal impairment	82 (65.6%)
Sustained seizure control achieved	8 (6.4%)

Seizures were documented in 121 (96.8%) patients during the course of illness and constituted the presenting manifestation in 72 (57.6%). In the remaining patients, seizures developed later as the disease progressed. Myoclonic seizures were the most frequent seizure type, observed in 66 (52.8%) children. These were typically brief, repetitive, and often stimulus-sensitive, involving axial, facial, or limb musculature. In several cases, myoclonic jerks occurred in clusters and showed a clear temporal association with EEG discharges. The detailed seizure types found in the study population are presented in Table 2.

Table 2. Distribution of Seizure Types in Children with SSPE

Seizure type	Frequency n (%)
Myoclonic seizures	66 (52.8%)
Movement disorder–associated paroxysmal events	27 (21.6%)
Generalized tonic–clonic seizures	17 (13.6%)
Atonic seizures	7 (5.6%)
Focal seizures	5 (4.0%)
No seizures	3 (2.4%)

The mean seizure frequency was 3.4 ± 1.6 episodes per day. Seizure burden varied considerably, with some patients experiencing multiple daily events leading to significant functional impairment. During ictal episodes, varying degrees of impaired consciousness were noted, ranging from brief alteration of awareness to complete unresponsiveness. Loss of postural tone and falls were documented in patients with atonic seizures, while prolonged recovery phases were common following generalized tonic–clonic seizures.

Movement disorders were identified in 27 (21.6%) patients and included dystonic posturing, choreoathetoid movements, and complex hyperkinetic phenomena. These events frequently coexisted with myoclonic seizures and were difficult to classify clinically, particularly in advanced disease stages.

EEG was performed in all patients and revealed characteristic periodic high-amplitude slow-wave complexes in every case. In the majority of patients, these periodic discharges were temporally correlated with clinical myoclonic jerks. Background activity showed progressive slowing in patients with longer disease duration, and an increase in the frequency and regularity of periodic complexes was observed in advanced stages. Postictal impairment was severe in 82 (65.6%) patients, manifesting as prolonged confusion, mutism, reduced responsiveness, or transient unresponsiveness lasting several minutes to hours. Multiple antiseizure medications were prescribed, most commonly valproate, benzodiazepines, and levetiracetam, either alone or in combination. Sustained seizure control was achieved in only 8 (6.4%) patients, while the majority continued to experience frequent seizures despite treatment.

DISCUSSION

This study describes the seizure spectrum in children with SSPE presenting to a tertiary-care center in Pakistan and highlights the substantial epileptic burden associated with this condition. In our clinical setting, seizures were not only common but frequently represented the initial neurological manifestation, leading to diagnostic consideration of SSPE.

The predominance of myoclonic seizures observed in this cohort supports the concept of SSPE as a progressive myoclonic epileptic encephalopathy; however, the presence of multiple seizure types in many patients reflects the evolving and diffuse nature of the underlying neurodegenerative process. Consistent with our findings, Khan et al. demonstrated that myoclonic seizures represent the predominant clinical manifestation in SSPE, reinforcing its characterization as a progressive myoclonic epileptic encephalopathy; the coexistence of multiple seizure types further reflects the diffuse and evolving nature of the disease process.[14]

More than half of the children presented with myoclonic jerks, which were often frequent and disabling. In many cases, these seizures constituted the initial clinical manifestation, emphasizing the importance of considering SSPE in the differential diagnosis of new-onset myoclonus in children, particularly in measles-endemic regions such as Pakistan.

An important observation in this cohort was the frequent occurrence of movement disorder–related paroxysmal events, including dystonia and complex hyperkinetic movements. In routine practice, these events are often difficult to classify and may be misinterpreted as non-epileptic phenomena, particularly in advanced disease stages. Recognition of these manifestations as part of the SSPE seizure spectrum is essential to avoid diagnostic delay and inappropriate management.

In agreement with previously published literature, including reports in Global Pediatric Health, the neuropathological involvement of basal ganglia and subcortical structures in SSPE provides a plausible explanation for the coexistence of myoclonic seizures with hyperkinetic and dystonic manifestations.[15] This overlap reflects the diffuse and progressive nature of measles virus-induced neurodegeneration. Moreover, consistent with global data, the occurrence of diverse seizure types—such as generalized tonic–clonic, atonic, and focal seizures—underscores the evolving electroclinical profile of SSPE, supporting the view that SSPE-related epilepsy represents a dynamic rather than static epileptic process.[16]

Our electroclinical findings are broadly consistent with those reported by Praveen-Kumar et al., who demonstrated characteristic periodic, bilaterally

synchronous high-amplitude slow-wave complexes on EEG in children with SSPE, often correlating with clinical myoclonus.[17] In our cohort, a similar pattern was observed, with generalized periodic complexes uniformly present and frequently time-locked with myoclonic jerks, reinforcing the concept of SSPE as a distinct electroclinical entity.

Progressive background slowing observed in patients with advanced disease likely reflects widespread cortical dysfunction and has been associated with poor neurological outcomes.[18] Furthermore, EEG abnormalities may provide insights into disease severity and progression. Progressive background slowing and increasing regularity of periodic discharges have been associated with advancing disease stages and poorer neurological outcomes in previous studies. Thus, EEG not only supports diagnosis but may also aid in clinical staging and prognostication.[19] In resource-limited settings, where access to advanced diagnostic modalities may be constrained, EEG remains a crucial and cost-effective tool for early diagnosis and disease staging. The poor response to antiseizure medications observed in this cohort underscores the pharmacoresistant nature of epilepsy in SSPE. Despite the use of commonly prescribed agents such as valproate, benzodiazepines, and levetiracetam, sustained seizure control was achieved in only a small proportion of patients. These findings are consistent with existing evidence that antiepileptic therapy in SSPE is largely palliative and does not alter disease progression.[19] This highlights the need for realistic counseling of families regarding therapeutic expectations, prognosis, and the emphasis on supportive and multidisciplinary care. Ketogenic diet or Low Glycemic Index Diet was not routinely implemented due to resource and compliance constraints; however, emerging evidence suggests potential symptomatic benefit in refractory cases.[20]

From a public health perspective, SSPE remains a tragic but preventable consequence of inadequate measles immunization. The vaccination gaps observed in this cohort reflect broader national challenges related to immunization coverage, vaccine hesitancy, and health system limitations. Strengthening routine immunization programs and improving public awareness remain the only effective strategies to eliminate SSPE and its associated neurological morbidity.

An important observation in this cohort was the frequent occurrence of movement disorder–related epileptic events, including dystonia and complex hyperkinetic movements. In routine practice, these events are often difficult to classify and may be misinterpreted as non-epileptic phenomena, particularly in advanced disease stages. Recognition of these manifestations as part of the SSPE seizure spectrum is essential to avoid diagnostic delay and inappropriate management.

This study has certain limitations. Its retrospective design may have resulted in incomplete documentation of seizure characteristics and treatment response. Being a single-center study, the findings may not be fully generalizable to all regions of Pakistan. Additionally, detailed staging data and long-term follow-up outcomes were not uniformly available. Despite these limitations, the relatively large sample size and systematic electroclinical assessment provide meaningful insights into the seizure spectrum of pediatric SSPE.

CONCLUSION

Children with SSPE show a characteristic but changing pattern of seizures, most commonly myoclonic and movement disorder–associated paroxysmal events in the form of dystonia, choreoathetoid movements and stereotyped hyperkinetic movements, with typical EEG findings. These seizures are usually frequent and difficult to control and add considerably to the neurological disability seen in affected children. Future multicenter studies incorporating longitudinal follow-up may further clarify the relationship between seizure patterns, electroencephalographic evolution, and clinical staging in SSPE.

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Faisal Zafar; Data collection, manuscript writing
Muhammad Tariq Aziz; Data Analysis, Manuscript writing
Arif Zulqarnain; data collection, data analysis, manuscript writing
Aysha Fayyaz; data analysis, manuscript writing
Amir Hanif; data interpretation, manuscript revision

All the authors have approved the final version to be published and agree to be accountable for all aspects of the work.

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