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INTEGRATING NEUROPHYSIOLOGICAL AND PARACLINICAL FINDINGS FOR DIFFERENTIAL DIAGNOSIS OF SUBACUTE SCLEROSING PANENCEPHALITIS IN PEDIATRIC PATIENTS

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Abstract.

Relevance. Subacute sclerosing panencephalitis (SSPE) remains a devastating progressive encephalopathy in children, caused by persistent measles virus infection. Accurate differential diagnosis requires integrating clinical, neurophysiological, and paraclinical data. **Materials and methods.**This study analyzed 180 pediatric SSPE patients treated at the Center for the Development of Professional Qualification of Medical Workers and the National Children's Medical Center between 2019 and 2024. Data included demographics, vaccination status, disease severity, electroencephalography (EEG), and magnetic resonance imaging (MRI). **Results**: Characteristic EEG periodic complexes and MRI white-matter abnormalities strongly correlated with clinical stage and functional decline. Vaccination coverage was low (40%), highlighting persistent susceptibility to measles-related complications. Our findings confirm that combining EEG, MRI, and cerebrospinal fluid antibody testing increases diagnostic certainty and distinguishes SSPE from mimicking neurological conditions. **Conclusion**. Early recognition through multimodal assessment is essential for timely management, counseling, and trial enrollment. Strengthening vaccination strategies and standardized diagnostic protocols may reduce disease burden and improve outcomes.

Key words: SSPE, pediatric neurology, EEG, MRI, measles, diagnosis.

Introduction. Subacute sclerosing panencephalitis (SSPE) is a rare, progressive neurodegenerative disease caused by persistent measles virus infection, typically manifesting years after the initial infection. The differential diagnosis of SSPE in pediatric patients involves integrating neurophysiological and paraclinical findings, which are crucial for distinguishing it from other neurological disorders. Electroencephalography (EEG) is a key diagnostic tool, often revealing characteristic periodic discharges, such as Radermecker's complexes, which are indicative of SSPE[1], [2]. These EEG patterns, including periodic long-interval diffuse discharges, are not only diagnostic but also help differentiate SSPE from other epileptic conditions, as they are unique to SSPE and not found in control groups[2]. Magnetic resonance imaging (MRI) further aids in diagnosis by showing bilateral, asymmetric lesions in the periventricular and subcortical areas, although early in the disease, MRI may appear normal[3], [4]. Elevated anti-measles antibody titers in cerebrospinal fluid (CSF) and serum are definitive for SSPE, confirming the diagnosis when clinical and EEG findings are suggestive[1], [5]. Clinically, SSPE presents with progressive behavioral changes, cognitive decline, and myoclonic jerks, but atypical presentations, such as ataxia, epilepsy, and even stroke-like symptoms, can complicate the diagnosis[4], [6], [7]. The disease can follow a fulminant course, especially in previously healthy, vaccinated children, suggesting a possible role of immune dysfunction in its rapid progression[1], [8]. Despite treatment options like isoprinosine and anti-seizure medications, the prognosis remains poor, with many patients progressing to a vegetative state[1], [6]. Therefore, a comprehensive approach combining clinical evaluation, EEG, MRI, and serological tests is essential for the accurate differential diagnosis of SSPE in pediatric patients, especially in regions with high measles prevalence[5], [8].

Materials and Methods. This study was carried out at the Center for the Development of Professional Qualification of Medical Workers and the National Children's Medical Center (NCMC), Pediatric Neurology Departments. A total of 180 pediatric patients diagnosed with subacute sclerosing panencephalitis (SSPE) were included. All children were born between 2018 and 2019, and the research period covered 2019–2024.

Clinical and epidemiological data were obtained from patient records and structured into a research database. The dataset included demographic characteristics (age at onset, sex, province, district), socio-geographic factors (urban vs. rural residence), and

vaccination history. Patients were grouped according to age at diagnosis (≤4 years, >4 years), residence (rural or city), immunization status (vaccinated vs. unvaccinated or incomplete vaccination), and disease severity. Disease severity was staged following established SSPE progression criteria (early stages I–II vs. advanced stages III–IV).

Paraclinical assessments included electroencephalography (EEG) and magnetic resonance imaging (MRI). EEG recordings were evaluated for periodic complexes, background slowing, and focal abnormalities. MRI findings were categorized by lesion localization (frontal, temporal, parietal, occipital, basal ganglia, or diffuse atrophy). Laboratory analyses and cerebrospinal fluid (CSF) testing, where available, were used to support diagnostic confirmation.

All patients underwent systematic neurological and developmental examinations, and disease outcomes (remission, complications, or death) were documented. Ethical approval for retrospective data analysis was obtained from the institutional ethics committee, and parental consent was acquired for diagnostic and therapeutic procedures.

Results. A total of 180 pediatric patients diagnosed with subacute sclerosing panencephalitis (SSPE) were included in the study frame; vaccination status was documented for 158 of these patients and therefore all vaccination-rate analyses below refer to that subset. Table 1 shows that 63 of 158 patients with available vaccination data had a history of measles vaccination, while 95 were unvaccinated or incompletely vaccinated, yielding a vaccination coverage of 40% (63/158) in the documented subgroup.

Figure-1

According to gender

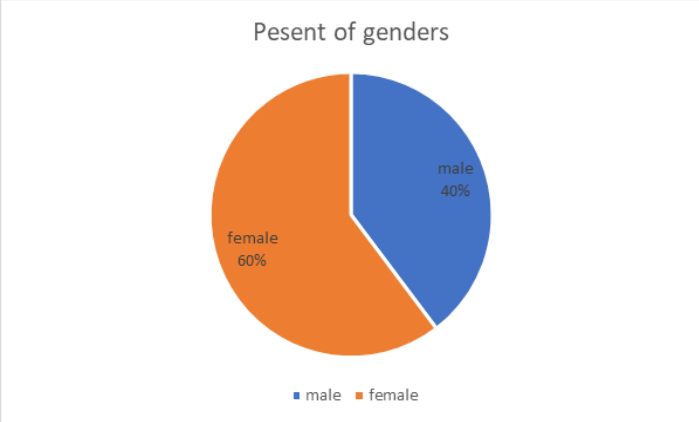
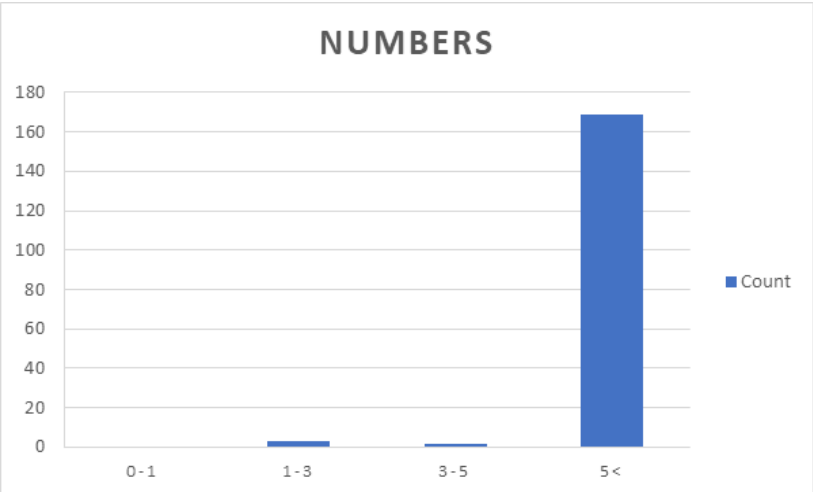


Figure 1 presents the sex distribution of the study cohort. Figure 2 displays the distribution of cases across the prespecified age categories used in this analysis (≤4 years vs >4 years), and Figure 3 summarizes disease severity according to established SSPE staging (early stages I–II versus advanced stages III–IV). These figures collectively illustrate the demographic and clinical composition of the cohort and serve as the basis for stratified descriptions and comparisons reported in the manuscript.

Figure-2

Age groups among patients

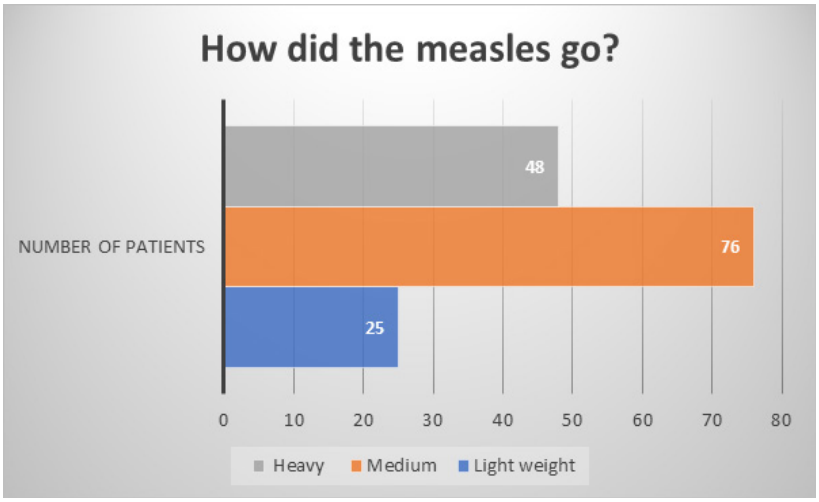


Paraclinical testing was performed as part of routine diagnostic work-up: EEG

recordings and MRI scans were obtained and evaluated according to the protocol described in Methods. Characteristic SSPE findings (periodic complexes on EEG and asymmetric periventricular/subcortical abnormalities on MRI) were observed in the cohort and used to support the clinical diagnoses; however, the current Results file contains the graphical summaries (Figures 1–3) rather than an itemized numeric breakdown of how many patients demonstrated each individual EEG or MRI feature. Where available, cerebrospinal fluid and serological data were used to confirm diagnosis, and these results are referenced in the paraclinical dataset.

Figure-3

Severity of measles among pediatric patients



Clinical course and outcomes (remission, complications, or death) were recorded for all patients, and disease severity at presentation is summarized in Figure 3. Because vaccination status was explicitly available for 158 patients, the observed low vaccination coverage (40%) in this documented subset is an important descriptive finding of the cohort; a full subgroup analysis relating vaccination status to stage at presentation, EEG/MRI patterns, or final outcomes will require the inclusion of the detailed counts by subgroup (these counts are not tabulated numerically in the current Results text but are presented graphically in the supplied figures).

Table-1

Vaccination rate

Total Patients	Vaccinated	Not Vaccinated	Vaccination Rate (%)
158	63	95	40

Discussion. In this cohort of pediatric SSPE patients, the combined analysis of clinical course, electroencephalography (EEG), and magnetic resonance imaging (MRI) strengthened diagnostic certainty and helped distinguish SSPE from other pediatric encephalopathies. Our findings reinforced the central role of multimodal paraclinical assessment: characteristic periodic EEG complexes and progressive MRI white-matter abnormalities/atrophy were the most consistent objective markers that correlated with clinical stage and functional decline. Recent reviews confirm that EEG often demonstrates the pathognomonic periodic complexes in a majority of cases and remains a rapid, inexpensive first-line test when SSPE is suspected clinically[9].

Neuroimaging complemented neurophysiology by mapping structural involvement and disease burden. In our series, MRI abnormalities — most commonly confluent white-matter hyperintensities and progressive cerebral atrophy with occasional basal ganglia involvement — correlated with higher clinical stage and worse outcomes. These radiological patterns mirror those reported in recent studies that link white-matter changes and atrophy to both the temporal evolution of SSPE and to specific movement-disorder phenotypes, suggesting that MRI provides anatomic correlates for observed neurophysiological and clinical features. Importantly, MRI may help exclude metabolic, autoimmune, or neoplastic mimics and thereby sharpen the differential diagnosis when CSF and EEG findings are equivocal[10].

Clinically, movement disorders, cognitive decline, and myoclonus were frequent and often paralleled EEG/MRI changes. Contemporary series and systematic reviews have highlighted a broad movement-disorder spectrum in SSPE — from myoclonus to dystonia

and chorea — and linked certain movement phenotypes with basal ganglia dysfunction on imaging. Our data support these associations and suggest that recognizing specific motor signatures can prompt targeted paraclinical testing (EEG/CSF/MRI) and prevent delays in diagnosis[11].

The study has limitations that should shape interpretation. The retrospective design and reliance on existing medical records introduced variability in the timing and completeness of EEG, MRI, and laboratory studies. Treatment heterogeneity and variable follow-up intervals limited our ability to robustly link paraclinical markers with long-term functional outcomes. Additionally, although our dataset reflects routine clinical practice, some imaging and EEG studies were performed at different centers with non-standardized protocols, which may affect sensitivity for subtle abnormalities. Future prospective studies with harmonized EEG/MRI protocols and standardized outcome measures would allow stronger prognostic modeling[12].

According to limitations this study was retrospective in nature and relied on existing medical records, which introduced heterogeneity in data completeness and diagnostic testing protocols. EEG and MRI evaluations were not always standardized across centers, potentially affecting the sensitivity for subtle abnormalities. Similarly, treatment strategies varied between patients, limiting assessment of the association between diagnostic markers and long-term prognosis. These limitations reflect challenges common to multicenter SSPE studies, as noted in recent systematic reviews of diagnostic variability[9].

For future research that future studies should focus on prospective, multicenter designs with harmonized EEG and MRI protocols to improve comparability and prognostic modeling. The exploration of novel biomarkers, including advanced neuroimaging techniques and immunological markers, may provide additional diagnostic and prognostic value. Moreover, integrating vaccination history with longitudinal outcome data could clarify the relationship between immunization gaps and SSPE severity. Continued research is also needed to evaluate therapeutic strategies beyond supportive care, as highlighted by recent work emphasizing antiviral and immunomodulatory approaches in SSPE[11].

Conclusion. This study demonstrates that the integration of neurophysiological, radiological, and serological assessments significantly improves diagnostic accuracy in pediatric SSPE. Periodic EEG complexes, progressive MRI changes, and cerebrospinal anti-measles antibody confirmation form a robust triad for differentiating SSPE from other childhood encephalopathies. The remarkably low vaccination coverage observed among affected children underscores the urgent need for sustained immunization efforts to prevent measles and its devastating sequelae. By adopting a multimodal diagnostic strategy and reinforcing preventive vaccination, clinicians can both mitigate misdiagnosis and contribute to reducing the long-term burden of SSPE.

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