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SUBACUTE SCLEROSING PANENCEPHALITIS IN A 24-MONTH-OLD CHILD: A RARE EARLY ONSET AND ALARMING PRESENTATION

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ABSTRACT

Subacute sclerosing panencephalitis (SSPE) is a rare, progressive, and fatal neurodegenerative disorder caused by persistent measles virus infection. Early-onset SSPE in children under five years is uncommon but often rapidly progressive. We report a 24-month-old unvaccinated male who presented with progressive myoclonic seizures, gait difficulty, and rapid neurodevelopmental regression. Electroencephalography (EEG) showed periodic generalized discharges, and CSF measles antibody titers were elevated, fulfilling modified Dyken criteria. The child was classified as stage III SSPE. Symptomatic therapy including anti-seizure medications and supportive care, along with a planned ketogenic diet, was initiated but neurological outcome remained poor. This case emphasizes the aggressive nature of early-onset SSPE and the importance of measles vaccination.

Keywords: Subacute sclerosing panencephalitis, Early-onset SSPE, Myoclonic seizures, Measles

INTRODUCTION

Subacute sclerosing panencephalitis (SSPE) is a chronic, progressive inflammatory encephalopathy caused by persistent measles virus infection. Typically, it manifests years after measles infection in older children and adolescents, leading to cognitive decline, myoclonic seizures, motor impairment, and death.[1]

Early-onset SSPE, defined as onset before five years, is rare but often rapidly progressive, especially in regions with suboptimal measles vaccination coverage.[2] Re-emergence of SSPE has been reported in populations with declining vaccination rates, highlighting the ongoing global relevance of this preventable disease.

This case describes a 24-month-old child who presented with a typical history of SSPE and was subsequently diagnosed with SSPE. This marks an unusual age of presentation of SSPE.

CASE PRESENTATION

A 24-month-old male presented to Lahore General Hospital with progressive myoclonic seizures, gait difficulty, and neurodevelopmental regression. He was born to non-consanguineous parents with uneventful birth. He was unvaccinated and had normal development until onset. Past history was significant for having developed a maculopapular rash at two months of age, which was diagnosed as measles infection. The diagnosis of measles at that time was made clinically by a pediatrician based on a typical maculopapular rash with associated prodromal symptoms. However, serological confirmation (measles IgM) was not performed, which is acknowledged as a limitation.

Regarding the current illness, onset occurred five months prior with frequent myoclonic seizures leading to falls. The myoclonic jerks were unresponsive to multiple anti-seizure medications. Over next three months, gait difficulty worsened, and within the last month, the child lost previously acquired milestones — walking, sitting, neck holding, speech, cognition, and responsiveness. No prior history of fever, trauma, or toxin exposure was present. Family history was unremarkable for any other sibling affected, highlighting the acquired nature of disease.

On examination weight was 11 kg and occipitofrontal circumference (OFC) was 47 cm, both corresponding approximately to the 15th–50th centiles according to WHO growth standards. Neurological findings included an encephalopathic child with frequent myoclonic jerks, more pronounced on awakening, with normal muscle tone, power ≥3/5 all limbs, DTRs 2+ bilaterally, extensor plantar responses, intact cranial nerves and no extrapyramidal signs. There were no dysmorphic features, skin lesions or organomegaly. Ophthalmological examination was also normal. Based on myoclonic jerks and regression, differential diagnosis of Subacute Sclerosing Panencephalitis, progressive myoclonic epilepsy and neuronal ceroid lipofuscinosis were considered and investigations were carried out accordingly (Table 1).

Table 1: Investigations of the patient	
Electroencephalogram	Periodic burst of generalized slow waves discharges followed by suppression (Consistent with SSPE)
Cerebrospinal fluid analysis	Cells: <5 cells/ul Protein: 36 mg/dL Glucose: 50 mg/dL Measles IgG: positive (qualitatively)
Metabolic workup	Plasma lactate: 14mg/dL Arterial blood gas: pH: 7.34 Bicarbonate: 20 mEq/L
Ophthalmological findings	No disc pallor or retinitis pigmentosa

His EEG showing burst suppression pattern is shown in Figure 1.

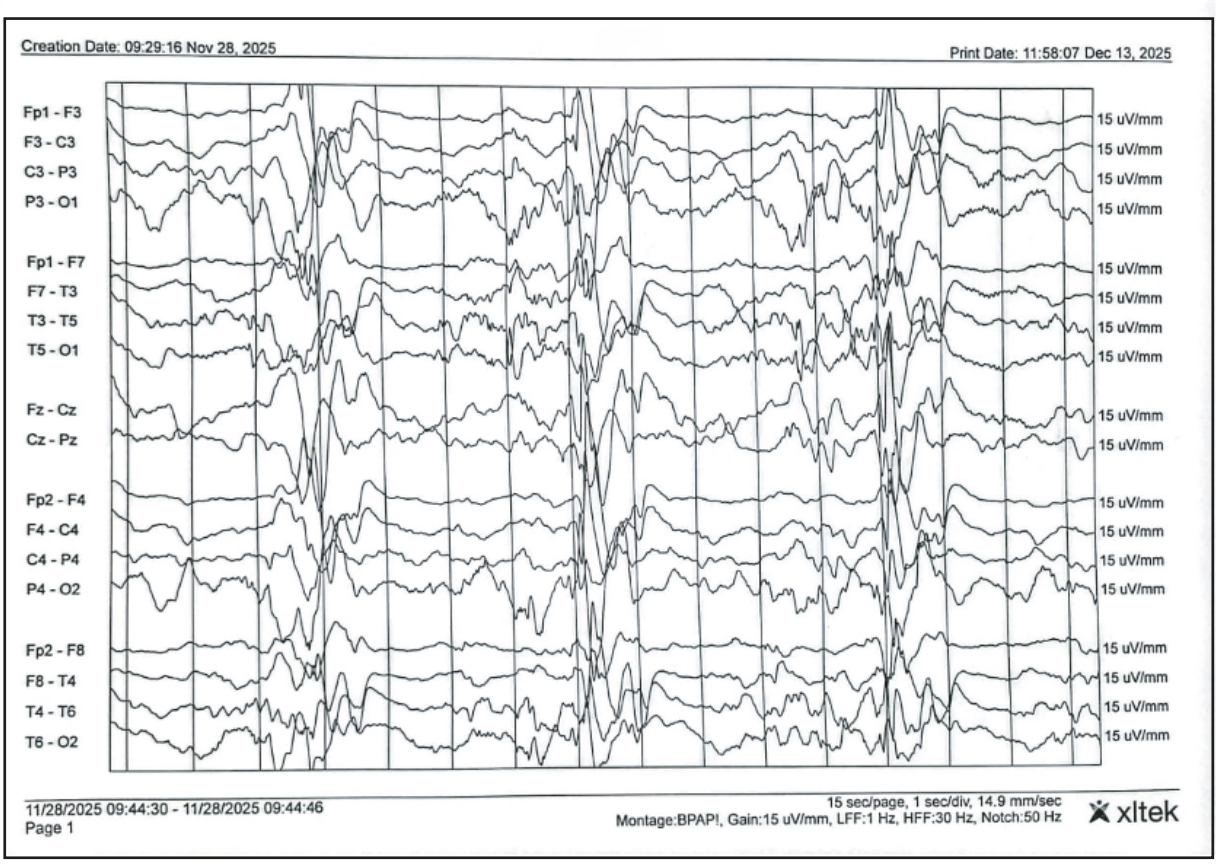

Figure 1: Electroencephalogram of the patient

Although measles infection in early infancy was not serologically confirmed, diagnosis of SSPE was made based on modified Dyken criteria (clinical features + EEG + elevated CSF measles antibodies). The child was classified as Stage III SSPE due to severe neuro-regression, inability to ambulate, altered consciousness, and frequent myoclonic seizures.
The child was managed with anti-seizure medication including levetiracetam, benzodiazepines and sodium valproate. As an adjunct therapy, ketogenic diet for seizure control was also initiated, along with supportive care including nasogastric feeding and physical rehabilitation. Anti-viral therapy was not initiated due to advanced stage and limited evidence of efficacy. Seizure frequency decreased modestly, but overall neurological status remained severely impaired. Parents were counseled regarding poor prognosis and the importance of vaccination for future prevention in their other kids.

DISCUSSION

SSPE is caused by persistent measles virus infection. While classically presenting years after infection, early-onset SSPE (<5 years) is rare but often aggressive. [2]

In our patient, fulminant measles infection in infancy likely triggered early-onset SSPE, leading to rapid neuro-regression, frequent myoclonic seizures, and gait difficulties. Early measles infection is a recognized risk factor for aggressive SSPE with shorter latency. A case series from the UK reported six children with SSPE, all older than two years, with a mean age of five years. Most had measles infection during infancy and experienced an aggressive disease course with severe neurological morbidity within six months of disease onset. [2]

The diagnosis of SSPE is based on the modified Dyken criteria, which require the presence of two major criteria and one minor criterion.[3] In our patient, the presentation was typical, with myoclonic seizures. CSF anti-measles antibodies were positive, fulfilling two major criteria. Among the minor criteria, EEG showed characteristic periodic generalized discharges consistent with SSPE.[4] Rapid loss of milestones and neurological symptoms justified stage III classification. Metabolic and genetic etiologies were considered; however, normal plasma lactate, normal arterial blood gas parameters, and absence of ophthalmological abnormalities made mitochondrial and neurodegenerative disorders such as neuronal ceroid lipofuscinosis less likely. Genetic testing could not be performed due to financial constraints.

Management remains largely supportive. Anticonvulsants and anti-inflammatory therapy were used. In our patient, ketogenic diet was initiated; however, its efficacy could not be adequately assessed due to poor compliance and short duration of follow-up (approximately one month). The patient remained clinically static during this period and was subsequently lost to follow-up. Ketogenic diet is emerging as a promising option for the management of SSPE and is currently being tried in these patients. It has anti-inflammatory and neuroprotective effects and may help slow or even reverse some of these neurodegenerative changes. Its benefits are thought to result from reduced oxidative stress, improved mitochondrial function, and suppression of pro-apoptotic (cell death–promoting) factors. It may help control myoclonic jerks and improve the cognitive and physical decline associated with SSPE.[5]

Other investigational therapies such as oral Isoprinosine (inosiplex), interferon-α, or combination therapy have been studied; a multicenter study by Gascon et al. (2003) found no statistically significant difference in long-term neurological outcomes between inosiplex alone and combined therapy, though both improved stabilization rates compared to historical controls. [6]

Prognosis in early-onset SSPE is poor, particularly in advanced stages; with rapid neurodegeneration and high mortality.[7] In patients with SSPE disease course is usually progressive, with only 5% undergoing spontaneous remission, while 95% usually die within five years.[8] This case emphasizes the aggressive course of early-onset SSPE and the importance of measles vaccination in prevention. [9,10]

CONCLUSION

SSPE should be considered in young children presenting with progressive myoclonic seizures and neurodevelopmental regression, especially in unvaccinated populations. Early-onset SSPE has a poor prognosis, reinforcing the critical role of timely measles immunization. Symptomatic management remains the mainstay, with adjuncts like the ketogenic diet for seizure control.

REFERENCES

1. Wendorf KA, Winter K, Zipprich J, Schechter R, Hacker JK, Preas C, et al. Subacute sclerosing panencephalitis: the devastating measles complication that might be more common than previously estimated. Clin Infect Dis. 2017 Jul 15;65(2):226-32.
2. Lam T, Ranjan R, Newark K, Surana S, Bhangu N, Lazenbury A, et al. A recent surge of fulminant and early onset subacute sclerosing panencephalitis (SSPE) in the United Kingdom: An emergence in a time of measles. Eur J Paediatr Neurol. 2021 Sep 1;34:43-9.
3. Rocke Z, Belyayeva M. Subacute Sclerosing Panencephalitis. 2023 May 19. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan.
4. Ugli GK. Characteristic eeg patterns in sspe and their correlation with clinical stages. Oriental J Med Nat Sci. 2025 mar 7;2(2):26-30.
5. Gutierrez J, Issacson RS, Koppel BS. Subacute sclerosing panencephalitis: an update. Dev Med Child Neurol. 2010 Oct;52(10):901-7.
6. Gascon GG. Randomized treatment study of inosiplex versus combined inosiplex and intraventricular interferon-α in subacute sclerosing panencephalitis (SSPE): International multicenter study. J Child Neurol. 2003 Dec;18(12):819-27.
7. Campbell H, Andrews N, Brown KE, Miller E. Review of the effect of measles vaccination on the epidemiology of SSPE. Int J Epidemiol. 2007 Dec 1;36(6):1334-48.
8. Nathan J, Kale DK, Naik VD, Bailur S. Substantial remission in subacute sclerosing panencephalitis by following the ketogenic diet: a case report. Cureus. 2019 Aug 25;11(8).
9. Garg RK, Mahadevan A, Malhotra HS, Rizvi I, Kumar N, Uniyal R. Subacute sclerosing panencephalitis. Rev Med Virol. 2019 Sep;29(5):e2058
10. Mubbashir Z, Tharwani ZH, Kambar T, Munawar S, Raphael O, Siddiqui I, et al. Subacute sclerosing panencephalitis: impact on public health, current insights, and future perspectives. Brain Behav. 2025 Feb;15(2):e70292.

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Arshad Mehmood: Concept, data collection, data analysis, manuscript writing.
Javeria Raza Alvi: Data interpretation, manuscript writing.
Sameen Qureshi: Concept, manuscript review
Ahmed Bilal: Data collection, manuscript review
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Tipu Sultan: Data interpretation, manuscript review
All the authors have approved the final version of the article and agree to be accountable for all aspects of the work.