CASE STUDY

94. Subacute Sclerosing Panencephalitis Presenting as a Stroke Mimic: A Case Report

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Background: Subacute sclerosing panencephalitis (SSPE) is a rare, progressive neurodegenerative disorder caused by persistent measles virus infection in the central nervous system. In SSPE, mutant forms of measles virus persist within neurons and glial cells, evading immune clearance. This results in chronic inflammation and progressive neuronal degeneration. The condition typically presents 6–8 years after the initial measles infection with cognitive decline, behavioral changes, and myoclonus, and later progresses to motor decline, seizures, and a vegetative state.

The estimated incidence of SSPE is 4–11 cases per 100,000 measles infections, though higher rates have been reported in lower- and middle-income regions, particularly when measles occurs at a young age. Although the global incidence has decreased with vaccination, SSPE continues to occur in areas with suboptimal measles immunization.

We present a case of SSPE in an unvaccinated young adult, initially suspected to have ischemic stroke.

The Case: A 22-year-old male, unvaccinated against measles, presented with acute neurological deficits, characterized by sudden-onset Right-sided Hemiplegia. Initial MRI showed acute cortical infarcts in the left fronto-parietal region. He received stroke-directed therapy, but there was no neurological improvement.

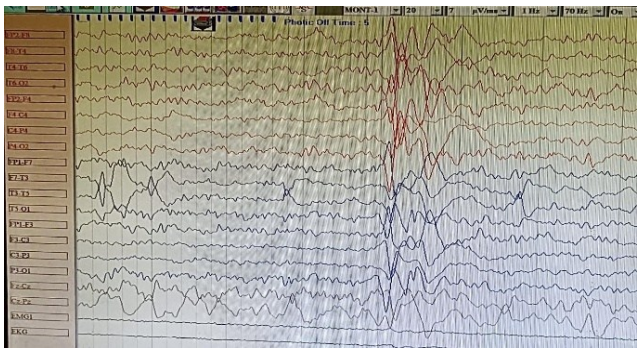
Over the following days, the patient developed progressive behavioral disturbances, irritability, and memory impairment, followed by myoclonic jerks. A repeat MRI one month later revealed diffuse T2/FLAIR hyperintensities involving the bilateral posterior parietal lobes, left frontal and temporal lobes, and left centrum semiovale, extending beyond the initial infarct-like lesions. These findings were more suggestive of an evolving encephalitic process rather than a vascular event.

Electroencephalography (EEG) demonstrated periodic epileptiform discharges (Figure 1). Cerebrospinal fluid (CSF) analysis showed markedly elevated measles-specific IgG antibodies (29.51 NTU; positive >11.0) with negative IgM, consistent with measles-related encephalitis. The diagnosis of SSPE was established in accordance with Dyken's criteria, fulfilling two major (clinical progression and elevated CSF anti-measles antibody titres) and one minor (EEG showing periodic epileptiform discharges) criterion.

The patient was given supportive treatment along with Isoprinosine, but despite these efforts, his condition continued to deteriorate, and he eventually succumbed.

Conclusion: This case highlights the rare presentation of SSPE with acute neurological deficits initially suspected as stroke in a young, unvaccinated male. The progression from an apparent vascular event to features of encephalitis shows the diagnostic complexity of SSPE and the need for clinicians to maintain a broad differential diagnosis in young patients with evolving neurological symptoms. Despite supportive measures and trial therapies, the prognosis remains poor, and strengthening measles vaccination coverage remains the only effective strategy to prevent this devastating disease.

Figure 1. EEG showing periodic epileptiform changes suggestive of SSPE



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