



Psychiatric Manifestations of Subacute Sclerosing Panencephalitis

Sana Dhamija, Suprakash Chaudhury* and Daniel Saldanha

Department of Psychiatry, Dr D Y Patil Medical College, Hospital and Research Centre, Dr D Y Patil Vidyapeeth, Pimpri, Pune

***Corresponding Author:** Suprakash Chaudhury, Department of Psychiatry, Dr D Y Patil Medical College, Hospital and Research Centre, Dr D Y Patil Vidyapeeth, Pimpri, Pune.

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Subacute sclerosing panencephalitis is a primary progressive chronic neurological disorder associated with demyelination that affects one in a million people in the West, but despite the availability of a widespread measles vaccination, it affects 21 people per million in developing nations like India [1,2]. Measles is primarily an infection that affects children between the ages of one and two. In contrast, SSPE is a long-term side effect that develops from a primary measles infection in a specific group of children and usually manifests six to eight years after the initial infection. This is the mutant measles virus's latent phase, during which it multiplies in neural cells by eluding humoral protection. The development of SSPE is indicative of a malfunctioning cell-mediated immune response during the initial measles infection, which leads to the development of an early humoral immunity and promotes intraneuronal infection [2].

Decline in cognition, certain behavioral issues, and myoclonic jerks or seizures that appear as gait issues and scholastic challenges are typically the first signs of SSPE (though these can be hard to recognize in the early stages). Four stages are used in some literature to categorize clinical presentations. The majority of stage 1 symptoms are psychological in nature. Unlike earlier stages, this makes it difficult to identify the symptoms of first stage of SSPE based on physical examination findings [2]. During this first stage, psychiatric manifestations precede the neurological manifestations of seizures, behavioural problems, and other focal neurological symptoms. However, these psychiatric manifestations are documented less in the literature [2].

Due to this, SSPE may go undiagnosed for several months, which frequently calls for a mental health referral. It is quite challenging to diagnose it in its early phases when there are no motor symptoms or indicators. This is especially true when the condition presents or begins atypically as a serious mental disease [3].

The latency period for SSPE can last up to three decades, according to published research, but symptoms often appear four to ten years after measles exposure [4]. Progressive intellectual decline, personality changes, myoclonus, ataxia, epileptic convulsions, and visual abnormalities are typically the first signs of illness [5].

The patient's age affects how they appear, and in some cases, an abnormal presentation-such as psychosis, catatonia, mania, or depression-delays the diagnosis in adulthood. The literature only has a small number of cases where psychosis or catatonia was the primary complaint [6].

Periodic discharges are seen on electroencephalography (EEG). Periventricular T2/FLAIR white matter abnormalities are seen on brain imaging. Significant brain atrophy is a hallmark of the advanced stage of SSPE. The presence of increased measles antibody titers in the cerebrospinal fluid (CSF) is required for a conclusive diagnosis [7]. Psychiatric symptoms, including as manic episodes, psychoses, catatonia, and delusions, have occasionally been documented in a large number of patients with SSPE [8]. Currently, the only information on the mental symptoms of subacute sclerosing

panencephalitis (SSPE) that is accessible is in the form of individual case reports. A systematic review analysed thirty published papers involving thirty-two patients. 17.9 years was the average age. Of the cases, 63% (20/32) had one of the three most prevalent mental presentations: schizophrenia, catatonia, or poorly defined psychotic disorders. Four patients showed signs of catatonia. Of the 32 cases, 22% (7 instances) had reports of depression, mania, and affective disorders. About 81% (26/32) of the individuals had an acute fulminant course of SSPE. SSPE rarely presents as psychiatric illnesses, which might result in incorrect diagnoses. Eventually, all these patients develop periodic myoclonus and encephalopathy [9]. The progressive loss of brain tissue brought on by a persistent measles virus infection may be the source of the psychotic symptoms in SSPE. According to one study, those with ongoing schizophrenia exhibited noticeably greater blood levels of the measles antibody titers than people without a history of mental illness [10]. A systematic review in 2024 found 16% of SSPE patients who presented with a mental presentation had catatonia [9]. Furthermore, the exact pathophysiology of catatonia in SSPE remains unclear. The frontostriatal network, which controls motor and behavioral activities in the brain, is thought to malfunction in cases of catatonia. The frontostriatal circuitry's basal ganglia are another target of the SSPE virus. Damage to the basal ganglia can interfere with this circuitry's regular operation, resulting in the motor and behavioral impairments that are typical of catatonia. The limbic system, which controls emotions, is the target of the measles virus. Depression may result from disruption of the brain circuits responsible for regulating mood brought on by damage to the limbic system. The pathophysiology of depression in SSPE may involve changes in neurotransmitter and immune system function in addition to direct brain tissue injury [11-13].

When a young person presents with acquired neurological impairments, SSPE should always be taken into consideration as a differential diagnosis. It's possible that SSPE is the great masquerader of our time. EEG may provide crucial hints, thus it is important to take this into account as soon as unusual neurological disorders in pediatric patients are being evaluated. The current case series aims to draw attention to SSPE's less common presenting manifestations [14].

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