ISSN 2394 - 2053 (Print)
ISSN 2394 - 2061 (Online)
RN: ASSENG/2016/70661 (RNI)
www.ojpas.com

CASE STUDY Open Journal of Psychiatry & Allied Sciences

A case report on subacute sclerosing panencephalitis (SSPE) from an Indian tertiary care system

Abstract

A prolonged measles virus infection is the cause of the unusual slow-progressing degeneration of the central nervous system known as subacute sclerosing panencephalitis (SSPE). This illness usually manifests in the early stages of adolescence, progresses slowly, and has a dismal prognosis. Usually, there is a six to eight years latent interval between measles infection and SSPE. It progresses gradually and finally results in death. The measles virus may induce an aberrant immune response that results in inflammation of the brain and this serious disease. Because SSPE is uncommon and sometimes misunderstood, it can be exceedingly challenging to diagnose. To arrive at a diagnosis, a complete examination and a full history are required. Here we have presented a case of 12 years old girl with SSPE who visited a tertiary care hospital in Assam, India.

Keywords: Diagnosis, measles virus, immune response.

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Received: 8 December 2024
Revised: 10 December 2025
Accepted: 10 December 2025
Epub: 15 December 2025

INTRODUCTION

Persistent measles infection is the cause of subacute sclerosing panencephalitis (SSPE), a slowly progressing neurological illness. This condition has a significant fatality rate, progresses slowly, and usually manifests in early adolescence.[1] It is brought on by inflammation and brain deterioration, which impairs cognitive and motor abilities.

Pathogenesis

The measles virus, a single-stranded ribonucleic acid (RNA) virus belonging to the Paramyxoviridae family, is the cause of SSPE. Complications can be both short-term and long-term. Adults over 20 years and children under five years exhibit higher mortality rates.[2,3] New study indicates that the pathogenesis has been attributed to both the virus' reproduction and host factors. Previous studies linked it to the virus' inability to make the measles M protein, while more recent studies link it to changes in the gene that codes for this protein. It has been suggested that reduced cellular immunity and limiting the virus to intracellular multiplication by certain antibodies are host characteristics that contribute to the sickness. Evidence suggests that patients with SSPE have an increased humoral immune response and a decreased cellular immunological response.[4,5]

Brain biopsy despite being the gold standard for the diagnosis, clinicians mostly use the Dyken's Criteria for it.

CASE REPORT

A 12-year-old girl, hailing from a rural background, belonging to a lower socioeconomic status, student of class VII, visited psychiatry outpatient department (OPD), Silchar Medical College Hospital (SMCH), Silchar, Assam, India presenting with memory loss, intellectual deterioration, and unexplained sudden decline in her scholastic performance.

The girl was apparently well about three months prior when her mother noticed that the child had started having difficulty in memorising her schoolwork and began to take much longer time to finish the assignments. She would sit down with her homework and would fiddle with the pen only to leave the pages blank. There were also multiple complaints from the school regarding her falling grades. It was completely in contrary to her earlier academic performance.

It was associated with decreased interaction and she showed little inclination for talking to her parents and friends. Most of the days she skipped her school only to be seen remain confined within the room. When friends and relatives came over to meet her, she only gave the response with a smile. It was also seen that she was having difficulty in recognising her friends.

Few weeks after, she started developing progressive weakness over the left side of her body and drooping of the head along with an unsteady gait. Earlier her mother thought the swaying of her body to one side was due to her fear of

getting punishment but gradually it was seen that there was also difficulty in holding objects and she needed support to walk. There were progressive gait disturbances. With time, she became more withdrawn and looked confused all the times. She also developed some abnormal stereotyped movements. She was seen to be making fists, fixing her hair or smelling her fingers. The patient was admitted in psychiatry ward, SMCH for further evaluation and treatment.

Her physical examination revealed no abnormality but on central nervous system examination, tone and power was reduced in right lower limb. On mental status examination, eye to eye contact was inadequate, psychomotor activity retarded, speech was monosyllabic, with decreased tone and flow but coherent and relevant. Her affect was shallow, restricted to lower side, stable and appropriate with no hallucinatory behaviour suggestive of perceptual disturbances. She was conscious, alert with comprehension intact but confused in left-right orientation and delay in following commands.

On day two of admission, she had two episodes of urinary incontinence. When asked for some response the patient only showed a brief hand movement. On day three of admission, she complained of slight blurriness of vision.

Her routine blood investigations and computed tomography (CT) scan brain reports were normal. Magnetic resonance imaging (MRI) brain showed T2/fluid attenuated inversion recovery (FLAIR) hyperintensity with gyral swelling involving cortical and subcortical white matter of right parieto-occipital region with diffuse restriction suggestive of acute infarct (Figure 1). Her electroencephalogram (EEG) showed characteristic bilateral periodic complexes (Figures 2 and 3). Her cerebrospinal fluid (CSF) analysis for measles immunoglobulin G (IgG) antibodies showed high titres of CSF total IgG and high CSF/serum quotient and significant high titres of serum total IgG CSF/serum quotient 2.6 (Table 1).

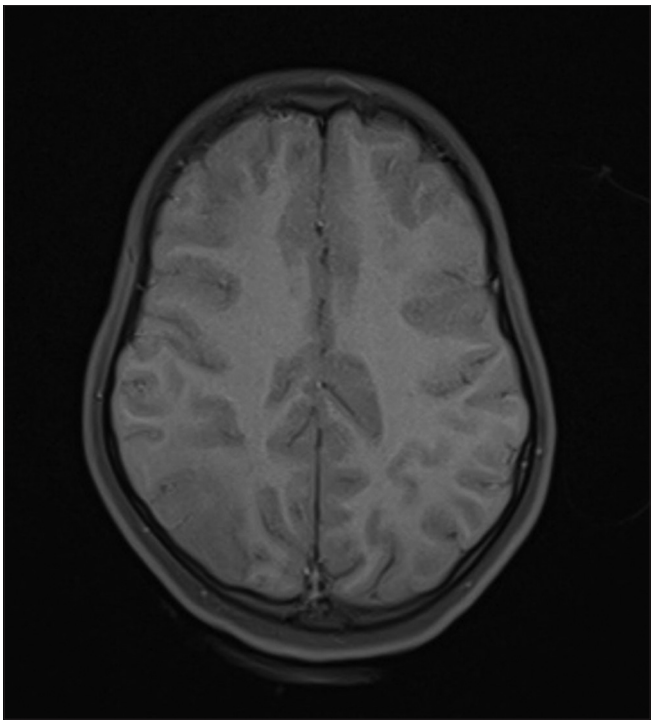

Figure 1: Magnetic resonance imaging (MRI) brain of the patient showing acute infarct involving parieto-occipital lobe on the right side.

Table 1: Cerebrospinal fluid (CSF) analysis of the patient showing high titres of measles specific immunoglobulin G (IgG) antibodies.

Serum IgG measles	437.78
CSF IgG measles	488.87
Serum total IgG	1673
CSF total IgG	45.5
CSF/Serum quotient	2.6

References- CFQ ref normal <1.3, CSFQ ref equivocal 1.3-1.5, CSFQ ref positive >1.5

Treatment given

Before the diagnosis was finally made the patient was put on tablet brivaracetam 50mg in two divided doses, tablet benfotiamine 100mg one tablet once daily, tablet clobazam 10mg in two divided doses, and syrup L-carnosine was given. After the final diagnosis the patient was eventually referred to a neurology centre.

Prognosis

The mortality rate is quite high in the case of SSPE, about 95%, while the remaining cases undergo spontaneous remission.[4,6]

DISCUSSION

Our case puts emphasis over the fact that it is through proper, detailed, and thorough examination and finally necessary investigations with EEG, MRI brain, and CSF analysis we can eventually reach up to diagnosis. As the disease can mimic acute encephalopathy, therefore we should include SSPE on the list of differential diagnosis of acute encephalopathy. A negative history of fever with rash in the past, as in our case does not simply exclude the diagnosis of SSPE. When atypical symptoms like psychiatric symptoms, uncontrolled seizures, or extrapyramidal symptoms are present, SSPE shows a fulminant course. Getting infected before the age of two years, increased viral load, and coinfection with other viruses puts greater risk for fulminant, atypical course.[3,4]

Diagnostic criteria

Dyken's criteria are used for the diagnosis which include two major and four minor criteria. Out of these, two major and one minor criteria are needed for the final diagnosis.[7]

Major criteria

Possessing a typical or atypical presentation is one of the principal requirements. Acute, rapid, subacute, chronic progressive, or chronic relapsing-remitting are the categories used to describe the typical presentation. Seizures and a very unusual age of presentation fall into the category of being atypical.

An additional important criterion is elevated anti-measles antibodies which needs to be greater than or equal to 1:4 in the CSF or 1:256 in the serum.

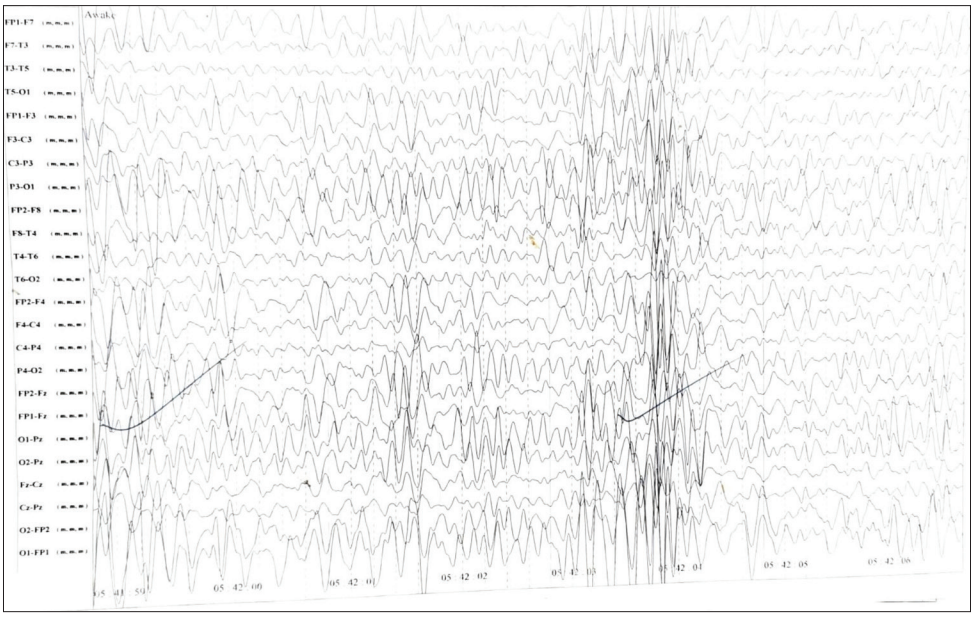

Figure 2: Electroencephalogram (EEG) of the patient showing bilateral periodic complexes.

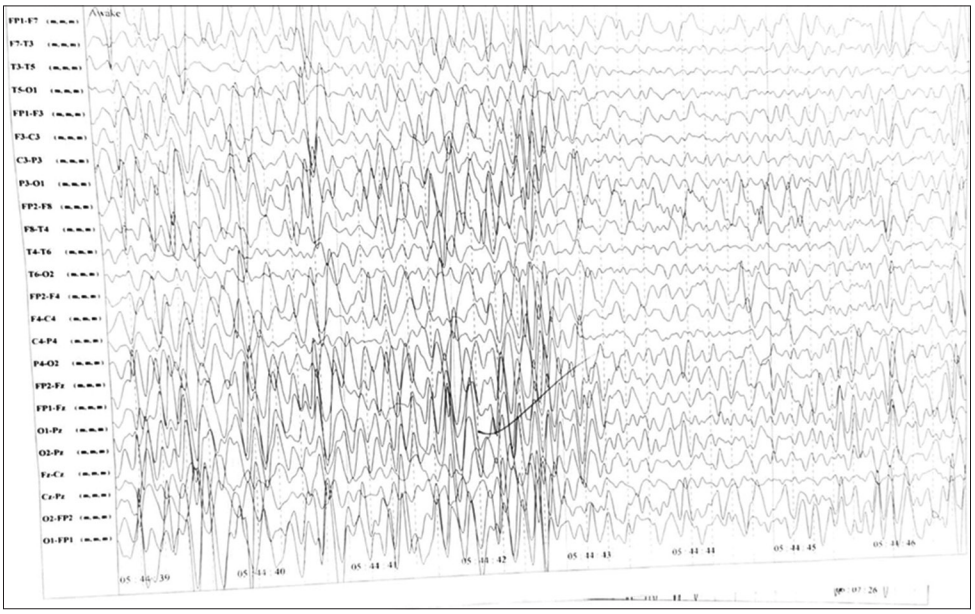

Figure 3: Electroencephalogram (EEG) of the patient showing more bilateral periodic complexes.

Minor criteria

Electrocardiogram (ECG) results in line with bilateral, synchronous, high-amplitude slow waves that occur at regular intervals. These are referred to as Radermecker complexes or slow-wave complexes.

A higher than average concentration of globulin in the CSF, which accounts for over 20% of the total protein in the fluid.

Measles virus detected in a brain biopsy.

Lastly, a molecular test that detects mutations in the genome of the wild strain of the measles virus.[7]

Progression of the disease

Stage I symptoms include personality or behavioural changes, such as irritability, dementia, lethargy, social disengagement, or speech regression, as the illness progresses.

Stage II: Progressive loss of motor function, characterised by dyskinesia, dystonia, and myoclonus.

Stage III includes spasticity, posturing, and extrapyramidal symptoms.

Stage IV: Autonomic failure, akinetic mutism, or transition into a vegetative state.[3]

Treatment

The current options try to slow the disease's progression, stabilise it, extend survival, or improve clinical outcomes. An oral antiviral that inhibits viral replication and functions as an immunomodulator is called isosine pranobex. Three doses of 100 mg/kg are given throughout the day, with a maximum dosage of 3000 mg. Isoprinosine and interferon-alpha (INF-alpha) are commonly used together. It is an immunomodulator that is injected intrathecally. Another nucleotide analogue that has been tried is ribavirin. When combined with INF-alpha, patients appeared to benefit only somewhat from it.[4,5] Numerous case reports suggest a ketogenic diet as an alternative treatment, as it has been shown to be neuroprotective.

Conclusion

Anti-measles antibodies should be checked in the CSF whenever there are odd clinical signs and a neuroimaging picture. Psychiatrists ought to be cognizant of the diverse manifestations of SSPE and ought to incorporate it into their differential diagnosis when a young child exhibits depressive symptoms and cognitive decline.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his/her consent for his/her images and other clinical information to be reported in the journal. The patient understands that his/her name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Sarma N, Das H. A case report on subacute sclerosing panencephalitis (SSPE) from an Indian tertiary care system. Open J Psychiatry Allied Sci. 2025 Dec 15. Epub ahead of print.

Source of support: Nil. **Declaration of interest:** None.