Subacute sclerosing panencephalitis (SSPE) is a slow-virus infection of the central nervous system which is caused by the measles virus. It is typically seen in children and adolescents; with the usual age of onset between 5-12 years. A large part of these patients have positive history for measles infection, usually at an age of less than two years. Measles infection contacted at less than one year of age carries a risk of SSPE, 16 times higher than that in children older than five years of age. The exact aetiology is not known; however it is postulated that an immature immune system fails to destroy the virus and the partially degraded virus persists in the central nervous system. The exact cause for re-activation of the dormant virus is not known. SSPE has also been reported in vaccinated children where it is thought to be sequelae of subclinical measles infection. SSPE is a slowly progressive neurodegenerative disorder which invariably has a fatal outcome.

The characteristic clinical presentation of SSPE is that of a previously well child presenting with regressive changes in intellect and personality. The parents often notice cognitive decline, impaired memory, decrease in scholastic
performance and behavioral changes in the child. Subsequently the child develops involuntary movements, such as myoclonic jerks and choreoathetosis. The limbs become rigid and spastic, eventually progressing to opisthotonus. Jabbour et al [3] classified the clinical course of SSPE into 4 progressive stages: Stage 1 patients have personality and/or behavioural changes; in stage 2, myoclonic jerks, seizures, and intellectual disabilities are noted; stage 3 is characterised by rigidity, spasticity and abnormal postures; and in stage 4, coma and death ensue. The natural history of SSPE can be extremely variable ranging from an awfully prolonged course, to almost an acute onset with rapid evolution of symptoms and death ensuing within a few weeks time. Autopsy reports and brain biopsies reveal inflammation of the meninges and the brain parenchyma with neuronal degeneration and gliosis. Demyelination is particularly observed in chronic cases. Advanced stages are associated with mild to moderate cerebral atrophy.

CT examination may be normal in the early stages. However, later bilateral symmetrical or asymmetrical hypPatenuating lesions in the cerebral white matter may be noted. Cerebral atrophy is evident in the advanced stages. MRI is a useful tool to assess the extent of cerebral involvement. MR-imaging scores superior to CT in detecting white matter abnormalities. In the early stages, MRI shows bilateral subcortical lesions which are often asymmetric. The parieto-occipital region is more frequently involved. Later confluent periventricular white matter changes become evident on T2-weighted and FLAIR images with encephalomalacia and cerebral atrophy. Involvement of the corpus callosum, brainstem, cerebellum, thalamus and the basal ganglia has also been reported. However, it has been widely accepted that often there is no correlation between the MR imaging findings and clinical stage of the disease. The diagnosis is usually established based upon the characteristic clinical picture, abnormal EEG findings and elevated titer of IgG measles antibody in the CSF and serum.

**Differential Diagnosis List:** Subacute sclerosing panencephalitis (SSPE)

**Final Diagnosis:** Subacute sclerosing panencephalitis (SSPE)

**References:**

Description: The ventricular system appears relatively prominent considering the patient’s age. Origin:
Description: Subtle symmetrical hypodensity is noted in bi-frontal white matter. Origin:
Description: Confluent and patchy areas of abnormal white matter signal are noted in the bilateral periventricular region. Origin:
Description: The ventricular system appears prominent for the patient’s age representing mild diffuse cerebral atrophy. Origin:
Description: Coronal FLAIR images reveal multifocal areas of subcortical white matter signal alteration.

Origin:
Description: Coronal FLAIR images reveal multifocal areas of subcortical white matter signal alteration.
Origin:
Description: Bi-frontal subcortical and deep white matter involvement is well depicted on these coronal FLAIR images. Origin:
Description: Bi-frontal subcortical and deep white matter involvement is well depicted on these coronal FLAIR images. Origin:
Description: Coronal FLAIR images show white matter signal alteration in left parieto-ocipital white matter. Origin:
Description: There is asymmetric involvement of the bilateral parieto-ocipital white matter as seen on this coronal FLAIR scan. Origin:
Description: Coronal FLAIR scan shows bi-temporal white matter hyperintensity. Origin:
Description: Bilateral temporal lobe white matter appears hyperintense on these axial T2-weighted image. Origin: