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Editorial

Jaw-opening myoclonus: A new semantic towards subacute sclerosing panencephalitis

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Subacute sclerosing panencephalitis (SSPE) is a relatively rare but essentially progressive neurodegenerative disease of the brain, caused by a chronic infection with a mutated measles virus in children and young adults.[1] Movement disorders (especially myoclonus of multiple patterns) are an implicate aspect of SSPE, besides cognitive decline, behavioural changes, seizures and motor disturbances.[2] Recently, a new phenomenology pattern has been identified and intricated in SSPE in the form of myoclonus of the jaw in association with background myoclonus in a subset of patients.

The initial proximate reports were observed in India (north), wherein Garg et al. observed recurrent involuntary opening of the mouth in a 6 year old girl with frequent falls and behavioural abnormalities of 3 months duration.[3] These jaw movements were frequent and periodic in the form of jaw-opening myoclonus (exhibiting a slow relaxation phase), accompanied by neck extension and myoclonic jerks involving the flexor muscles of the limbs (upper limbs more than lower limbs and trunk) which synchronized with Radermecker complexes in electroencephalogram (EEG). Its phenomenology probably represents the extension of the disease from the cortical/subcortical origin to the brainstem, with consequent myoclonus affecting the trigeminal-innervated jaw muscles, occurring in association with axial myoclonus. However, the magnetic resonance imaging (MRI) of brain was normal in the patient. The authors further stated that this type of clinical observation was not identified in their research on 50 patients of SSPE.

This was soon followed by a couplet report 4 months later by Pandey et al. who reported similar phenomenology in two patients (6-year-old-girl and 8-year-old-girl) having rapidly progressing cognitive decline associated with striking jaw-opening myoclonus, who were also diagnosed with SSPE.[4] The first patient exhibited quasi-periodic, sudden jerky movement of the jaw and right upper and lower limbs with slow relaxation, while the second had sudden episodes of jaw-opening myoclonus accompanied by eye-widening, giving a surprised look, followed by head and truncal-negative myoclonus leading to frequent falls. Brain imaging showed non-enhancing posterior-predominant symmetric white-matter T2/FLAIR hyperintensities with left fronto-parietal atrophy in first case, and T2/FLAIR hyperintensities in bilateral occipital white matter in the second case.

Subsequently, an observational SSPE study observed 7 patients (out of 56) demonstrating jaw-opening myoclonus.[5] Strikingly, majority (5 out of 7) were male, with an age range of 3–18 years, and median duration of symptoms 4 (1.5–24) months. JOM was synchronous with myoclonus in other regions in five patients and asynchronous in two, with one patient having a preceding type of JOM. They also proposed that Jaw-opening myoclonus may be a feature of advanced disease in SSPE. The brain MRI was essentially normal in 3 cases, generalized cerebral atrophy in 1, bilateral

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hippocampal signal changes in 1, discrete frontal white matter hyperintensity in 1, and asymmetrical frontoparietal white matter signal change, along with basal ganglia signal changes in last case.

In the recent case description by Madduruli et al., a prominent jaw-opening myoclonus was observed in a 15-year-old-girl of SSPE presenting with multifocal myoclonus, and recent onset of cognitive disturbances of 4-months duration.[6] Distinctively, her brain imaging (MRI) was unremarkable. Radermecker complexes were however present.

The authors have phenotypically pointed this distinct jaw-opening myoclonus in SSPE from India. The aggregate of 10 such cases have given newer insights into exploration of movement disorder in SSPE on a global stage. The clinical phenomenology, synchronicity, periodicity, duration, association and course of jaw-opening myoclonus in SSPE is extensively discussed through these case reports. The jaw-opening myoclonus in SSPE is now essentially described as slow myoclonus, hung myoclonus, epileptic spasm, or periodic dystonic myoclonus.[7] However, the topographical representation of this movement could not be ascertained with brain imaging as of now. The analysis of these SSPE cases points towards lacunae in structurally localizing the lesion network via present-day brain MRI testing. Modalities like FDG-PET MRI, and fMRI might be beneficial in its structural confirmation and provide newer insights.

Conflict of Interest

None.

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