Time lag In the recognition of absence seizures to diagnosis and treatment of childhood absence epilepsy in Singapore

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Background and Objective: Childhood absence epilepsy (CAE) occurs in children of school age, with strong genetic predisposition in otherwise normal children. CAE is characterized by very frequent absences i.e. abrupt impairment of consciousness, with cessation of voluntary activity. The electroencephalograph (EEG) shows bilateral, synchronous spike-waves, usually 3 Hz, on a normal background activity, which is often precipitated by hyperventilation. The absence seizures of CAE may be difficult to be recognized by either the parents or teachers. However, the very frequent absence seizures and electroencephalographic discharges may potentially affect the child's learning and education in school.[1] The objective of this study was to determine the time period from the onset of absence seizures to diagnosis and treatment for children with CAE in Singapore.

Methods: We reviewed the EEG and medical records of Singaporean children managed in our institution with the diagnosis of CAE from 1993 to 2003. The inclusion criteria were based on the definition by the ILAE[2] and the EEGs were verified to have findings consistent with CAE. We excluded the following which were incompatible with CAE: 1) Eyelid or perioral myoclonia, rhythmic massive limb jerking, and single or arrhythmic myoclonic jerks of the head, trunk, or limbs; 2) Mild or no impairment of consciousness during the 3-Hz discharges; 3) Photic and other sensory precipitation of clinical seizures; 4) Brief EEG 3-Hz spike-wave discharges of less than 4 seconds, multiple spikes (more than 3) or ictal discharge fragmentations

Results: We had 23 patients, 15 girls and 8 boys, with onset of absence seizures between 4 to 11 years of age. All the patients had typical 3-Hz spike-and-wave discharges lasting at least 4 seconds in their routine EEGs, with activation seen in all 21 children who could perform hyperventilation adequately. The average time period between the onset of seizures and the diagnosis was 10.5 months, with 57% of the patients being diagnosed at least 6 months after the onset of absence seizures. All were treated with valproic acid (VPA) except for one who had a possible drug allergy. Seventeen patients (77%) were seizure-free on low dose VPA (< 30 mg/kg/day). One patient was on high dose VPA (>30mg/kg/day), and 4 patients required two anti-epileptic drugs (VPA and ethosuximide) to achieve clinical response.

Conclusion: CAE is fairly easily treated using just low dose VPA monotherapy for the majority of our patients, as is the experience worldwide. However, our study showed a marked time lag in the recognition of absence seizures for children with CAE. As early diagnosis and appropriate treatment may be important to prevent adverse seizure, intellectual or psychosocial outcomes, a greater awareness of this condition among the medical and educational personnel in Singapore is beneficial.

References

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