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Dr Elsayed M. Ali graduated from the University of Khartoum (Sudan) in 1980, and completed his residency training in pediatrics at Suleimania Children Hospital, Riyadh; and later obtained the Membership and Fellowship of the Royal College of Physicians of Ireland.

He was appointed as Consultant Pediatrician at Suleimania Children Hospital and Clinical Assistant Professor, King Saud University, Riyadh between 1994 and 1998.

Dr. Ali was trained in pediatric neurology and neurophysiology at King Saud University, Riyadh and University College London (Great Ormond Street Children Hospital and Institute of Neurology, Queen Square). After that he joined King Fahd Military Medical Complex (KFMMC) in Dhahran as Consultant Pediatric Neurologist. Over the last 15 years, he actively participated in establishing the Child Neurology Service at KFMMC, which is a very busy service that also serves children referred from different military hospitals in the Eastern Province of Saudi Arabia.

Dr. Ali participated as guest speaker at many national and international neurology conferences, and he published articles in the field of pediatric neurology. His main areas of interest are epilepsy, neuromuscular disorders and movement disorders.

Abstract

Epileptic encephalopathies in early infancy with suppression-bursts EEG pattern in children from The Eastern Province of Saudi Arabia

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Background and Objectives:

Epileptic encephalopathy in early infancy with suppression-bursts EEG pattern comprises two distinct epileptic syndromes, early infantile epileptic encephalopathy (EIEE, or Ohtahara syndrome) and early myoclonic encephalopathy (EME). The aim of the present communication is to draw attention to these debilitating early infantile epileptic encephalopathies, so that they can be early recognized and appropriately treated.

Methods:

Chart records of 8 children (3 males and 5 females) admitted to child neurology service between September 1999 and August 2012 were reviewed. Digital video EEG was performed in 6 patients and paper EEG in 2. Neuroimaging and metabolic workup was done for all patients.

Results:

Five cases fulfilled criteria for early infantile epileptic encephalopathy (EIEE) and 3 cases for early myoclonic encephalopathy (EME). The mean age at the time of onset of seizures was 12 days. EEG in all patients showed suppression-burst (SB) patterns. In two cases it evolved into hypsarrhythmia. Seizure semiology consisted of tonic spasms in some cases, and generalized or partial patterns in others. MRI brain was abnormal in 5 cases (3 scans revealed cortical atrophy, 1 hemimegalencephaly, and 1 white matter demyelination). Metabolic workup was abnormal in 3 cases (high CSF/plasma glycine ratio in nonketotic hyperglycinemia, increased serum very long chain fatty acids (VLCFAs) in Zellweger syndrome and high serum lactate in a child with cytochrome c oxidase deficiency). Anticonvulsant therapy was successful initially in one patient, but the patient later relapsed. Another child was referred for hemispherectomy, whereas in the rest of patients (6 cases) various combinations of antiepileptic drugs were tried and have failed in controlling their seizures and halting the deterioration of psychomotor development. Unfortunately no genetic testing was done for the cryptogenic cases.

Conclusion:

Epileptic encephalopathies in early infancy need to be recognized early, thoroughly investigated to determine the underlying etiology, and aggressively treated to alter their poor prognosis

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