Neurology Asia 2004; 9 (Supplement 1) : 107 – 108

NEUROPHYSIOLOGY AND IMAGING

Periodic lateralized epileptiform discharges: Aetiology and association with EEG seizures

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Background and Objective: Periodic lateralized epileptiform discharges (PLEDs) are EEG abnormalities consisting of repetitive spike or sharp wave discharges, which are focal or lateralized over one hemisphere and that recur at almost fixed time intervals. When present bilaterally and independently, they have been termed BIPLEDs. PLEDs have most commonly been associated with cerebral infarctions but are also seen in other cerebral diseases such as encephalitis, tumour or demyelination. It still remains controversial whether PLEDs represent actual ictal activity, predispose to epileptic seizures or are simple indicators of underlying aetiology.[1-5] The objective of this study was to investigate the aetiology of PLEDs and the co-existence of EEG seizure activity.

Methods: A retrospective review of EEG reports in a tertiary hospital between 1998 – 2004 was conducted. The EEG recordings of reports with periodic lateralised or localised epileptiform discharges (PLEDs or BIPLEDs) were selected and analysed. Clinical data of these patients were also traced specifically for the results of imaging studies (MRI/CT brain), liver/renal function tests and cerebrospinal fluid (CSF) analyses. EEG seizures were defined when there were rhythmic EEG activity (PLEDs themselves &/or other concomitant activity) *with* progression in field, frequency &/or amplitude with or without clinical seizure correlate.

Results: Forty-five of 7,500 patients (0.6%) had EEG criteria fulfilling PLEDs (37 PLEDs; 8 BIPLEDs). The mean age was 66 years (range, 26-92), with 21 males and 24 females. The aetiologies corresponding to the PLEDs were cerebrovascular accident (53%), central nervous system infection (16%), encephalomalacia (7%), tumour (7%), demyelination (2%), metabolic (2%) and idiopathic (13%).

Eighteen patients (40%) had concomitant EEG seizures, all ipsilateral to the recorded PLEDs. Nine of these patients had cerebrovascular accident (7 ipsilateral, 2 contralateral). Two patients each had central nervous system infection, ipsilateral encephalomalacia/gliosis, ipsilateral tumour, and normal neuroimaging. No neuroimaging was found in 1 patient.

Mortality was encountered in 15 patients (33%), 1-50 days post-EEG. Eight patients had cerebrovascular accident, 3 had central nervous system infection, 2 had tumour, 1 had significant metabolic derangement and the remaining 1 had no discernible aetiology. Only 4 of 18 patients (22%) with concomitant EEG seizures died. Of those who died, 73% had PLEDs in >75% of their EEG. Mortality was almost 50% among those with >75% of PLEDs in their EEG versus 20% among those with <75% of PLEDs in their EEG.

Discussion and Conclusion: The majority of our patients with PLEDs had cerebrovascular disease in the same region. Concomitant EEG seizures in the region of PLEDs were recorded in over 1/3 of our patients. The EEG seizures suggest possible epileptogenic potential of PLEDs. Our patients had a high mortality rate (33%). A higher percentage of PLEDs was associated with higher mortality, perhaps representing greater severity of the underlying aetiology. Presence of EEG seizure was not associated with higher mortality.

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