Background

Periodic lateralized epileptiform discharges (PLEDs) and Periodic Epileptiform Discharges (PEDs) are seen with a variety of neurological conditions. They may be seen in the acute state and sometimes represent an ictal pattern (1). The significance of PLEDS and PEDs to predict prognosis may in part be due to limited amount of EEG in these patients. We sought to observe patterns of evolution and localization of these patterns. We reviewed patients with PEDs during continuous EEG to ascertain the relationship of mortality to etiology and persistence of PEDs.

Methods

This was a retrospective study reviewing records of consecutive adult patients undergoing continuous EEG (cEEG) at Wake Forest University Baptist Medical Center, Winston-Salem, North Carolina between March, 2008 to September, 2009. Charts were reviewed to identify patterns seen on EEG, diagnosis, duration of cEEG monitoring, and mortality. Periodic epileptiform discharges were further classified into generalized PEDs and bilateral PLEDS. Patient outcomes and EEGs were reviewed and analyzed both during and after an acute illness.

Results

Number of Patients and Duration of Monitoring

Eleven patients out of total of one hundred sixty-six were found to have PEDs pattern on EEG accounting for a total of seven percent of patients. The duration of monitoring for these patients ranged from 1-14 days with a mean of 4.09 days.

PLEDs Features

The predominant EEG pattern in patients who suffered a global insult consisted of BiPLEDs. The patient with alcohol withdrawal seizures was also found to have focal T2/FLAIR changes on MRI which was related to prolonged seizure activity. PLEDS were treated as an ictal pattern where clinical improvement was accompanied by EEG improvement.

Results (cont)

Outcomes of patients with PLEDS pattern

Sixty-four percent of patients who were found to have PEDs died. In those who survived and had focal PLEDS, large vessel stroke was the cause. The etiologies in those who did not survive consisted of anoxic brain injury (42%), sepsis (42%), and bacterial meningitis (14%). (Table 1). All patients were placed on anti-epileptic medications at the time of initiation of cEEG. In eight patients, PEDs were considered to be electrographic seizures on their cEEGs. (Table 2).

Discussion

The overall prevalence in other case series have identified PLEDS in 0.8% of patients (3). Conditions that are associated with diffuse cerebral dysfunction in setting of periodic discharges on EEG have an increased mortality (6). Our group is different from other series because of factors such as absence of cases of brain tumor and chronic causes for the PLEDS. (2)

We also found that patients who survived the acute critical illness period were more likely to have focal neurologic injury. The EEG pattern in those who died consisted of either BiPLEDs or GPEDS (2). Clinical improvement appeared to be associated with EEG improvement which is different from some reported series.

Conclusion

Surviving patients were found to have focal structural insults as compared to a more diffuse brain involvement in those who died. Changes in EEG may accompany clinical improvement but the nature of insult was more predictive of outcome in this small group. Further prospective studies with larger sample sizes are needed in order to determine if the underlying etiology for PLEDS can clarify prognosis.

References