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NUS-07 Headache co-morbidity and seizure control in patients with epilepsy

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Introduction: Headache (HA) and epilepsy (EP) are common neurological diseases with significant morbidities and mortalities and pose enormous economic burden to our societies. They share clinical characteristic including episodic nature and response to anticonvulsants, and they potentially share similar pathophysiological mechanisms, for example, channelopathies. Here, we hypothesis that the presence of headache comorbidity in patients with epilepsy is associated with poor seizure control.
Methods: We studied 904 consecutive patients with seizure disorders in the Epilepsy clinic of Queen Mary Hospital. We successfully interviewed 474 patients with a previously validated, standardized questionnaire to screen for HA comorbidity. Patients with non-epileptic seizure, mental retardation or dementia were excluded. The HA diagnosis was subsequently validated by an independent neurologist who was unknown to individual EP diagnoses and the relevant investigation results. All patients were prospectively followed-up for 12 months to optimize their antiepileptic drugs (AEDs) therapy aiming at achieving a seizure free state. During which, HA diagnosis was also blinded from the attending neurologist for AEDs optimization. No specific treatment, other than simple analgesic, was prescribed for HA during the study period.
Results: HA comorbidity was found in 157 patients with EP. Among which, 61 of them could not achieve a seizure free state after a 12 months period. In contrast, only 68 of 315 epileptic patients without HA comorbidity failed to achieve a seizure free state. ($p< 0.000$, Odd ratio = 2.308, 95% Confidence interval 1.519 – 3.507).
Conclusions: Headache is a common co-morbidity for patients with epilepsy and associates with a poor seizure control state. This indicates that monitoring headache comorbidity of patients with epilepsy is equally important. Further study is needed to determine if modification of headache comorbidity could improve seizure control.
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NUS-08 Postictal psychosis related lateral temporal hyperperfusion

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Introduction: Postictal psychosis is a rare complication of epileptic seizure characterized by reversible psychotic symptoms after flurries of seizure attack. It was attributed to a phenomenon similar to Todd's paralysis without definite proof. We hypothesis regional hyperperfusion cerebral SPECT defect is associated with postictal psychosis complicating epileptic seizure. Two years ago, we reported our preliminary data of 99mTc-HMPAO cerebral SPECT findings in two patients with PIP. This study is an extension of our previous work.
Methods: We prospectively recruited patients with postictal psychosis and performed 99mTc-HMPAO SPECT scan during PIP. Interictal scans were performed at least 4 weeks apart from the PIP and taking off from anti-psychotic medications, if any. The average uptake ratios for each predefined brain region of interest (ROI) were normalized to cerebellum. Semi-quantitative analysis of normalized rCBF was performed and asymmetry index (ASI) was calculated during interictal state and during postictal psychosis.
Results: We identified six consecutive patients with PIP including two patients we reported previously. Statistically significant difference could be identified between interictal ASI and PIP ASI over lateral temporal region ($p=0.017$). No statistical significant difference could be observed between interictal ASI and PIP ASI for other ROIs. No statistical difference could be detected between the mean normalized count for each individual ROIs and no association could be detected between lateralization of seizure onset and hyperperfusion abnormalities.
Conclusions: This study provides further support to our earlier report of lateral temporal hyperperfusion in patients with postictal psychosis. Although hyperperfusion abnormality in SPECT can be found in Todd's paralysis, such findings are more commonly found in patients with cerebral hyperactivity conditions. Considering the clinical characteristics of postictal psychosis of preceded lucid interval and crescendo–decrescendo clinical course, our findings suggest an alternative pathogenic mechanism for the development of postictal psychosis, for example, activation of subcortical circuit. Furthermore, our observations may be related to the pathogenesis of force normalization.