Results: Amongst the thirteen patients with age ranging 14–44 years (mean 26.8 ± 9.2) 62% of them had daily seizures. MRI abnormalities were identified in 8 patients (62%), PET showed concordant findings in 7 patients (88%). When utilized, the mean duration of intracranial EEG recordings was 8.0 + 7.2 days (range 2–23 days). All patients underwent a primary motor cortex-sparing resection of the suspected epileptogenic cortex. The mean postoperative follow up period was 23.2 months (range 8–62 months). Twelve out of 13 (92%) were seizure free (Engel 1a) outcome at the last follow-up assessment; one patient with Engel 2a outcome at 28 months. Six patients (46%) had immediate new focal neurological deficits, however all six patients had recovered completely within three months.

Conclusion: The surgical strategy of a primary motor cortex-sparing resective surgery for perirolandic FCD is associated with an excellent early seizure-freedom rate and no permanent neurological deficits. Since the ultimate goal of resective epilepsy surgery is seizure freedom with simultaneous functional preservation, similar long term outcome studies should ultimately guide the resection strategy.

http://dx.doi.org/10.1016/j.ijep.2015.12.042

Endogenous kynurenic acid, a tryptophan metabolite, synthesis is altered in resected brain specimens obtained from patients with mesial temporal lobe epilepsy

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Mesial temporal lobe epilepsy (MTLE) is the most common form of drug-resistant epilepsy where hippocampus is responsible for unprovoked seizures. The hallmark of MTLE is enhanced glutamatergic excitatory neurotransmission. Kynurenic acid (KYNA), a tryptophan metabolite, is a specific inhibitor for NMDA type glutamate receptor. Animal and human epilepsy models have documented that concentration of KYNA and kynurenine pathway metabolites are altered in brain. Alteration in synthesis of endogenous KYNA may provide an insight in understanding of hyper activation of glutamate receptors in MTLE. Resected hippocampus tissues were obtained from MTLE patients. Slices (350 μm) were prepared and incubated with 100 μM kynurenine containing artificial cerebrospinal fluid (ACSF) for 2 h at 30 °C. KYNA was estimated using HPLC with dual wavelength fluorescent detection system. We observe that endogenous production of KYNA was significantly less in MTLE tissues (0.06433 μg/μl ± 0.019918) compared to non-epileptic controls (1.91995 μg/μl ± 0.63759). The result of present study suggests that decreased endogenous KYNA production could be a reason for hyperactive glutamatergic neurotransmission and targeting this pathway could act as a potential therapeutic target for MTLE.

Purpose: MTLE is the most common form of drug-resistant epilepsy where hippocampus is responsible for unprovoked seizures. The hallmark of MTLE is enhanced glutamatergic excitatory neurotransmission. Kynurenic acid (KYNA), a tryptophan metabolite, is a specific inhibitor for NMDA type glutamate receptor. It is synthesised and released from cortical astrocytes. In animal models of epilepsy and in human tuberous sclerosis complex it has been shown that concentration of KYNA and kynurenine pathway metabolites are altered in brain. Alteration in synthesis of endogenous kynurenic acid may provide an insight in understanding of hyper activation of glutamate receptors in MTLE.

Methods: Hippocampal tissue from drug resistant MTLE patients and tissues resected from the tumour margin during brain tumour surgery of seizure-free patients as non-epileptic control specimens were used for the study. Three hundred fifty micrometre thick slices were prepared from the tissues, followed by they were incubated with 100 μM kynurenine containing artificial cerebrospinal fluid (ACSF) for 2 h at 30 °C (Rozsa et al., 2008). Following incubation, the solutions were stored at −80 °C. KYNA was estimated using HPLC with dual wavelength fluorescent detection system (excitation 344 nm emission 404 nm) (Xiao et al., 2008).

Result and conclusion: Altered excitatory synaptic transmission is one of the primary causes of seizure generation in patients with mesial temporal lobe epilepsy (MTLE). The present study is designed to delineate the contribution of glutamatergic tone under resting conditions to the hyper excitability in patients with MTLE. Resected hippocampal tissues were obtained from patients with MTLE. In these samples spontaneous excitatory postsynaptic currents (EPSCs), sensitive to NMDA receptor antagonist APV (50 μM) and AMPA receptor antagonist CNQX (10 μM) were recorded from pyramidal neurons at −70 mV. We observed that frequency of EPSCs were 28.2% higher in slices obtained from patients with MTLE compared to that in case of non-epileptic controls. We also examined spontaneous fast current transients (CTs) recorded from these pyramidal neurons under cell-attached configuration. The frequency of CTs increased in the absence of extracellular Mg^{2+} in brain slice preparations and was completely blocked by APV. We found that the frequency of CTs in pyramidal neurons were higher in case of MTLE samples compared to non-epileptic controls. This study suggests that enhanced endogenous activity of NMDA receptor contributes to excitability in pyramidal neurons of slice preparations obtained from patients with MTLE.

http://dx.doi.org/10.1016/j.ijep.2015.12.043