Temporal lobe epilepsy

Mesial temporal sclerosis (MTS) is the most common cause of intractable temporal lobe epilepsy.

Classification

1. Anatomical Classification

Mesial Temporal Lobe Epilepsy (MTLE) – Originates in the hippocampus, amygdala, or parahippocampal gyrus. Lateral Temporal Lobe Epilepsy (LTLE) – Originates in the neocortex of the temporal lobe. 2. Etiological Classification

Structural – Hippocampal sclerosis, tumors, malformations, strokes, trauma. Genetic – Familial lateral temporal lobe epilepsy (e.g., LGI1 mutations).

Infectious – Post-encephalitic, neurocysticercosis. Metabolic – Rare metabolic disorders affecting the temporal lobe. Immune-mediated – Autoimmune encephalitis (e.g., anti-LGI1, anti-GAD65). Unknown – No identifiable cause. 3. Electroclinical Classification Focal Aware Seizures – Conscious, with auras (déjà vu, epigastric rising, auditory/olfactory hallucinations). Focal Impaired Awareness Seizures (FIAS) – Altered awareness with automatisms. Focal to Bilateral Tonic-Clonic Seizures – Progression to generalized seizures. 4. Syndromic Classification MTLE with Hippocampal Sclerosis (MTLE-HS) – Common, often drug-resistant. MTLE without HS – No hippocampal atrophy, variable drug response. Familial Lateral Temporal Lobe Epilepsy (ADLTE) – Genetic, linked to LGI1 mutations. Post-traumatic or Post-encephalitic TLE – Secondary to injury or infection. Autoimmune-associated TLE – Linked to limbic encephalitis.

Bilateral temporal lobe epilepsy

Bilateral temporal lobe epilepsy

Clinical features

Temporal lobe epilepsy (TLE) is a chronic neurological condition characterized by recurrent seizures (epilepsy) which originate in the temporal lobe with progressive neurological disabilities, including cognitive deficit, anxiety and depression.

The seizures involve sensory changes, for example smelling an unusual odour that is not there, and disturbance of memory.

Treatment

Temporal lobe epilepsy treatment

Mesial temporal lobe epilepsy

Mesial temporal lobe epilepsy.

Neocortical temporal lobe epilepsy

Neocortical temporal lobe epilepsy

Unilateral temporal lobe epilepsy

Unilateral temporal lobe epilepsy

Case series

Sixty patients with drug-resistant temporal lobe epilepsy who underwent anterior temporal lobectomy were enrolled. Anterior hippocampal samples were collected after surgery and analyzed by immunofluorescence (n = 7/group). They also evaluated the expression of HMGB1 in TLE patients with hippocampal sclerosis and measured the level of plasma HMGB1 by enzyme-linked immunosorbent assay. The results showed that 28.3% of the patients (17/60) had comorbid depression. HMGB1 was ubiquitously expressed in all subregions of the anterior hippocampus. The ratio of HMGB1-immunoreactive neurons and astrocytes was significantly increased in both TLE patients with hippocampal sclerosis and TLE patients with comorbid depression compared to patients with TLE only. The ratio of cytoplasmic to nuclear HMGB1-positive neurons in the hippocampus was higher in depressed patients with TLE than in non-depressed patients, which suggested that more HMGB1 translocated from the nucleus to the cytoplasm in the depressed group. There was no significant difference in the plasma level of HMGB1 among patients with TLE alone, TLE with hippocampal sclerosis, and TLE with comorbid depression. The results of the study revealed that the translocation of HMGB1 from the nucleus to the cytoplasm in hippocampal neurons may play a previously unrecognized role in the initiation and amplification of epilepsy and comorbid depression. The direct targeting of neural HMGB1 is a promising approach for anti-inflammatory therapy [1)]

Yang et al., therefore, examined all patients who underwent ANT-DBS or TL-RNS for drug-resistant TLE.

They performed a retrospective review of patients who were treated with either ANT-DBS or TL-RNS for drug-resistant TLE with at least 12 months of follow-up. Along with the clinical characteristics of each patient's epilepsy, seizure frequency was recorded throughout each patient's postoperative clinical course.

26 patients underwent ANT-DBS implantation, and 32 patients underwent TL-RNS for drug-resistant TLE. Epilepsy characteristics of both groups were similar. Patients who underwent ANT-DBS demonstrated a median seizure reduction of 58% at 12-15 months, compared to a median seizure

reduction of 70% at 12-15 months in patients treated with TL-RNS ($p > 0.05$). The responder rate (percentage of patients with a 50% decrease or more in seizure frequency) was 54% for ANT-DBS and 56% for TL-RNS ($p > 0.05$). Incidence of complications and stimulation-related side effects did not significantly differ between therapies.

They demonstrated in a single-center experience that patients with drug-resistant TLE benefit similarly from either ANT-DBS or TL-RNS. Selection of either ANT-DBS or TL-RNS may therefore depend more heavily on patient and provider preference, as each has unique capabilities and configurations. Future studies will consider subgroup analyses to determine if specific patients have greater seizure frequency reduction from one form of neuromodulation strategy over another [2].

1)
Li XL, Wang S, Tang CY, Ma HW, Cheng ZZ, Zhao M, Sun WJ, Wang XF, Wang MY, Li TF, Qi XL, Zhou J, Luan GM, Guan YG. Translocation of High Mobility Group Box 1 From the Nucleus to the Cytoplasm in Depressed Patients With Epilepsy. ASN Neuro. 2022 Jan-Dec;14:17590914221136662. doi: 10.1177/17590914221136662. PMID: 36383501.
2)
Yang JC, Bullinger KL, Dickey AS, Karakis I, Alwaki A, Cabaniss BT, Winkel D, Rodriguez-Ruiz A, Willie JT, Gross RE. Anterior Nucleus of the Thalamus Deep Brain Stimulation Versus Temporal Lobe Responsive Neurostimulation for Temporal Lobe Epilepsy. Epilepsia. 2022 Jun 15. doi: 10.1111/epi.17331. Epub ahead of print. PMID: 35704344.