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Case Report

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Electroclinical Study of Adult Epilepsy and The Damage of Epileptic Network

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Abstract

We have clinically analyzed 16 cases of adult epilepsy ranging from 19 to 70 years old. The seizure patterns found were tonic-clonic seizures and absence seizures. 9 cases were classified as generalized epilepsy and 7 cases as partial epilepsy. Neuroimages study revealed the following damage of brain parenchyma: hemisphere porencefalia and cists, temporal loss of neurons, gliosis, occipital cyst and occipital retraction of left ventricle, and calcifications in temporal and parietal region. The electroclinical findings were correlated with the epilepsy network examining the lesion of brain parenchyma induced by the seizure types. Frontotemporal, frontoparietal and occipital brain regions appeared as the most affected regions.

Introduction

In the elderly the clinical profile with regard to etiology, postictal conditions, and comorbidities clearly depends on the age of the patients and age of onset of epilepsy (Stefan et al., 2014). Epilepsy is a symptom complex with multiple risk factors and a strong genetic predisposition rather than a condition with a single expression and cause. Comorbidities are increasingly recognized as important aetiological and prognostic markers [1].

Adult-onset idiopathic generalized epilepsy (IGE) is a relatively frequent and benign disorder. Seizures are usually provoked and are easy to control. Patients in this age group may often be misdiagnosed as having non-lesioned partial epilepsy. Pedigree analysis suggests that adult-onset IGE, like classical IGE, has a genetic alethiology [2]. A clinical syndrome often has multiple possible genetic causes, and conversely, different mutations in one gene can lead to various epileptic syndromes. Most common epilepsies, however, are probably complex traits with environmental effects acting on inherited susceptibility, mediated by common variation in particular genes [3].

An epileptic seizure is defined as a sudden occurrence of transient signs and symptoms caused by abnormal and excessive or synchronous neuronal activity in the brain. Focal and generalized epilepsy are the two most frequent types of epilepsy; diagnosis is based on the type of seizures, and epilepsy syndrome, as well as the patient's age and sex, comorbidities, and potential drug interactions. In the present study we analyze the electroclinical features of generalized and partial epileptic types, their relationship with the damage of brain parenchyma or epileptic network and their comorbidities

Results

Case Reports

Case 1. NP. 25 years old. F. Patient product of twin births with complex partial epilepsy, right hemiparesis and psychomotor retardation. Febrile convulsive seizures at one year old and normal EEG. At seven years old presented frequent generalized seizures, deficit of learning and memory. NMR images showed left hemisphere porencephalia and cists. Patient treated with Valproic Acid and Carbamazepine.

Case 2. KA. 19 years old. F. Generalized convulsive crisis. EEG showed abnormal slow intermittent active waves from frontal and parietal regions. NMR showed normal brain images and ethmoidal sinusopathy. Patient treated with Carbamazepine.

Case 3. BH. 52 years old. F. Patient with absence type epileptic crisis, high blood pressure, anxiety and severe partial loss of weight. EEG 2019 showed slow waves in all brain regions. Patient treated with Carbamazepine.

Case 4. LB. 47 years old. F. Patient with generalized convulsive crisis and transitory loss of consciousness, dizziness, headache.

Aura with dizziness and tachycardia. Abnormal EEG 2013 showed moderate epileptic focal activity. EEG 2017 generalized outbreak of spikes and slow waves. Patient treated with Carbamazepine and Lamotrigine.

Case 5. FD. 19 years old. F. Patient with three generalized convulsive crisis and loss of consciousness with preceding strange auras at 13, 17 and 18 years old. Patient with family history of epilepsy. Genetic epilepsy. Interictal normal EEGs 2017 and 2018. Carbamazepine and Lamotrigine.

Case 6. 39 years old. F. Patient with focal convulsive syndrome, depression, insomnia and negative thinks. EEG 2016 showed irritated focus in right frontal- temporal regions. Received Valproic Acid and Risperidone.

Case 7. TV. 29 years old. F. Patient with generalized convulsive syndrome, loss of consciousness, loss of sphincter control in the last 1 year old. At 10 years old suffered intense and frequent convulsive crisis, two, four or six per day. EEG 2013 showed outbreak of acute slow waves in left temporal region. NMR images showed temporal loss of neurons, NMR 2015 showed a glioma frontal, gliosis and associated inflammatory process in right frontal region. The epileptic process was refractory to Valproic Acid, Carbamazepine. As a final treatment received Neurontin and Pregabalin. Surgical treatment was suggested to her family.

Case 8. AZ. 22 years old. F. Patient with partial epilepsy, hypoxic-ischemic encephalopathy and right hemiparesis due to mother dystocic delivery and bleeding. Hospitalized during 15 days. EEG showed disorganized slow way rhythm without evidence of irritative focus. NMR images showed occipital cyst and occipital retraction of left ventricle. Patient treated with Valproic acid.

Case 9. AK. 30 years old. M. Convulsive generalized syndrome since 10 years old. Generalized and frequent convulsive syndrome with aura featured by facial tics. EEG showed outbreak of 4 Hz Theta waves and Delta acute waves. NMR images normal. Ethmoidal inflammatory changes. Received Phenytoin and Valproic acid.

Case 10. EH. 25 years old. F. Patient with partial epilepsy. One year ago, suffered a brain trauma after fall with headache, right hemiparesis. And loss of weight. Depression, and sexual abuse one year ago. EEG showed outbreak of slow waves. Receive Valproic acid.

Case 11. AK. 30 years old. M. Convulsive generalized syndrome for 10 years old. Generalized and frequent convulsive syndrome with aura featured by facial tics. EEG outbreak of 4 Hz Theta waves and Delta acute waves. NMR showed normal brain images and ethmoidal sinusitis. Patient treated with Valproic Acid and Carbamazepine.

Case 12. NB. 49 years old. F. Brain trauma at 15 years old. Generalized convulsive syndrome 7 days after trauma. Patient with severe labor stress. EEGs 2004 and 2006 showed outbreak of acute waves in right and left brain parasagittal regions and multifocal epilepsy. Patient treated with Phenobarbital.

Case 13. LS. 56 years old. F. Convulsive syndrome Grand mal type since 2 years old years. Convulsive syndrome accentuated during menstrual periods, loss of weight. EEG showed paroxysmal discharges. TAC images showed calcifications in temporal and parietal regions. Refractory epilepsy to Phenobarbital, Valproic acid Lamotrigine.

Case 14. YC, 62 years old. F. Generalized convulsive syndrome. Loss of weight. EEG excessive slow waves. Moderate epileptiform activity. Patient treated with Carbamazepine.

Case 15. NS.70 years old. M. Patient with cerebral palsy and frequent focal convulsive syndromes of arms and legs. High blood pressure, diabetes, head deviation to the left and insomnia. EEG showed outbreak of generalized slow waves. Patient treated with Diphenylhydantoin, Carbamazepine and Valproic Acid.

Case 16. PS. 23 years old. M. Patient with frequent generalized convulsive syndrome Grand mal type since two years ago. EEG 2012 showed slow waves and paroxysmal activity. EEG 2017 showed outbreak of slow and acute waves in all brain regions. Patient treated with Diphenylhydantoin, Carbamazepine, and Valproic Acid.

Results

We have clinically analyzed 16 cases ranging from 19 to 70 years old. 12 cases were classified as generalized epilepsy and 4 cases as partial epilepsy. Some patients showed frontoparietal and parietotemporal epilepsy. The following comorbidities were found: High blood pressure, diabetes, anxiety, depression, dizziness, sleep disorders, language disturbances and loss of weight. Patients No. 7 and 14 showed refractory epilepsy. Neuroimages study revealed the following damage of brain parenchyma corresponding to the epileptic nertwork: hemisphere porencefalia and cysts, temporal loss of neurons, gliosis, occipital cyst with occipital retraction of left ventricle, and calcifications in temporal and parietal region.

Discussion

We have found 12 cases with generalized epilepsy. Idiopathic generalized epilepsies (IGE) are characterized by specific EEG changes including 3- to 5-Hz generalized spike-and-wave discharges. The thalamus and its cortical interactions are considered essential in the production and propagation of spike-and-wave discharges. In humans the involvement of the centromedian and parafascicular part of the corticoreticular system and the anterior nucleus part of limbic system has been found during generalized spike-and-wave discharges. The different time courses suggest that the posterior intralaminar nuclei may be involved in epileptic discharge initiation or early propagation, while the anterior nucleus may only play a role in its maintenance [4]. The typical seizure semiology for frontal lobe epilepsy (FLE) includes

unilateral clonic, tonic asymmetric or hypermotor seizures. Interictal electroencephalograms (EEG) usually reveal interictal epileptiform discharges and rhythmical midline theta, which has localizing value [5].

Generalized epileptic seizures are underlied by specific circuits where GABAergic synapses are involved at different levels. The role of these synapses depends on (i) the type of epilepsy and (ii) their localization within the central nervous system [6]. GABAergic synaptic inhibition, which is a critical regulator of neuronal excitability, is closely involved in epilepsy. Interestingly, fast GABAergic transmission mediated by Cl- permeable GABAA receptors can bi-directionally exert both seizure-suppressing and seizure-promoting actions. Accumulating evidence suggests that chloride plasticity, the driving force of GABAA receptor-mediated synaptic transmission, contributes to the double-edged role of GABAergic synapses in seizures [7].

The role of astrocytes in Epilepsy

Recent studies have implicated that astrocytes play important roles in physiology, but these cells also emerge as crucial actors in epilepsy. Astrocytes are abundantly coupled through gap junctions allowing them to redistribute elevated K(+) and transmitter concentrations from sites of enhanced neuronal activity. Investigation of specimens from patients with pharmacoresistant temporal lobe epilepsy and epilepsy models revealed alterations in expression, localization, and function of astroglial K(+) and water channels. In addition, malfunction of glutamate transporters and the astrocytic glutamate-converting enzyme, glutamine synthetase, has been observed in epileptic tissue. These findings suggest that dysfunctional astrocytes are crucial players in epilepsy and should be considered as promising targets for new therapeutic strategies [8].

The substantia nigra (SN) has been identified as a critical site at which GABA-agonist drugs act to reduce susceptibility to a number of types of experimentally induced generalized seizures. Because chemical or electrical stimulation of SN does not initiate convulsions, it appears that seizure activity generated elsewhere in the brain may be amplified or sustained by activity in these nigral outputs [9].

The Absence type Epilepsy

We have found one patients with absence type epilepsy (Case No 3).The mechanisms underlying absence SE are uncertain and may include both genetic and environmental factors. The termination of absence seizures has been hypothesized to be due to persistent activation of a depolarizing current in thalamocortical neurons that inactivates T-type calcium channels. SE could thus result from dysfunction of this channel or mechanisms that hyperpolarize thalamocortical neurons-these include decreased cortical inhibition, increased reticular thalamic neuronal activity or increased thalamocortical neuron GABA(B)-receptor activation [10].

Refractory Epilepsy

In cases 7 and 14 we observed refractory epilepsy. Refractory mesial temporal lobe epilepsy (MTLE) is the most frequent focal epilepsy and is often accompanied by deficits in social cognition including emotion recognition, theory of mind, and empathy. Consistent with the neuronal networks that are crucial for normal social-cognitive processing, these impairments have been associated with functional changes in fronto-temporal regions. Our results show that lower affective and cognitive empathy was associated with smaller volume in predominantly right fronto-limbic regions, including the right hippocampus, parahippocampal gyrus, thalamus, fusiform gyrus, inferior temporal gyrus, dorsomedial and dorsolateral prefrontal cortices, and in the bilateral midbrain [11].

Genetic Epilepsy

We have found some patients with a family history of epilepsy, suggesting a genetic aetiology. Novel insights into the genetic aetiology, comorbidities and prognosis of the genetic generalized epilepsy (GGE) syndromes have emerged and challenge traditional concepts about these conditions. Evidence has shown that the mode of inheritance in Genetic generalized epilepsy (GGE) is mostly polygenic. Genetic generalized epilepsy (GGE) syndromes start during childhood or adolescence, and four commonly persist into adulthood, making up 15-20% of all cases of epilepsy in adults Neuropsychological and imaging studies indicate similar abnormalities in unaffected relatives of patients with GGE, supporting the concept that underlying alterations in bilateral frontothalamocortical networks are genetically determined. Reduced cognitive functioning has been documented in the genetic generalized epilepsies (GGE). Among a number of hypothesized causal mechanisms, some evidence from other epilepsy syndromes suggests the impact of epileptiform discharges [12].

Epilepsy and Neurobehavioral Disorders

Some patients herein examined showed anxiety, depression, dizziness, sleep disorders, and language disturbances (Cases No. 1,3,4,6,9,11and16).The psychiatric disorders and associated poor psychosocial outcomes are recognized to be a common sequelae of epilepsy [13].

Epilepsy Induced By Severe Labor Stress And Hot Climate

In adult epilepsy we have observed late onset epileptic process induced by labor stress, or severe environmental factors, such as hot climate. Such cases had either a benign course or a refractory process.

Adult Onset Ideopathic Generalized Epilepsy

Adult onset ideopathic generalized epilepsy (IGE) is a relatively frequent and benign disorder. Seizures are usually provoked and are easy to control. Patients in this age group may often be misdiagnosed as having non-lesional partial epilepsy. Early postictal EEG and sleep deprivation studies may improve the detection of these patients. Pedigree analysis suggests that adult onset IGE, like classical IGE, has a genetic aetiology [2].

Conclusions

12 cases were classified as generalized epilepsy and 4 cases as partial epilepsy. Some patients showed frontoparietal and parietotemporal epilepsy. The following comorbidities were found: High blood pressure, diabetes, anxiety, depression, dizziness, sleep disorders, language disturbances and loss of weight. Patients No. 7 and 14 showed refractory epilepsy. Neuroimages study revealed the following damage of brain parenchyma corresponding to the epileptic nertwork: hemisphere porencefalia and cysts, temporal loss of neurons, gliosis, occipital cyst with occipital retraction of left ventricle, and calcifications in temporal and parietal region.

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