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Clinical and Diagnostic Significance of Glutamate in Patients with Focal Epilepsy

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Abstract Epilepsy is a widespread neurological disorder of the brain, the main clinical feature of which is a chronic predisposition to recurrent, sudden and unpredictable epileptic seizures. The obtained data indicate the possibility of using the level of glutamate in the blood serum as one of the diagnostic criteria for focal epilepsy. **The aim of the study** was to evaluate the diagnostic significance of changes in serum glutamate levels before and after taking Zonisamide in comparison with carbamazepine in patients with focal epilepsy. **Material and methods:** Twenty-six serum samples from patients diagnosed with focal epilepsy were analyzed. The examination included a clinical and neurological examination, laboratory blood tests (general and biochemical analysis), computed tomography (CT) or magnetic resonance imaging (MRI) of the brain, and electroencephalography (EEG). Glutamate concentrations in the serum were determined spectrophotometrically. **Results:** Increased brain electrical activity was found to be associated with elevated serum glutamate levels. The highest glutamate levels (19.2±8.51 ng/ml) were recorded in patients with focal epilepsy with impaired awareness and transition to bilateral tonic-clonic seizures. **Conclusions:** The obtained data indicate a strong correlation between serum glutamate levels and zonisamide intake. It is concluded that zonisamide intake at a dose of ≥300 mg per day can reduce seizure frequency by >50%.

Keywords Focal epilepsy, Glutamate, Spectrophotometry, Zonisamide, Carbamazepine

1. Introduction

More than 70 million people worldwide suffer from epilepsy, which is characterized by recurrent spontaneous seizures and is accompanied by numerous neurological, cognitive, and psychosocial consequences [1,6]. Currently, the main cause of epileptic seizures is believed to be a disturbance in excitation and inhibition processes in nervous tissue [2,4]. Excitation processes are realized through the glutamatergic system.

Glutamate is one of the main excitatory neurotransmitters in the brain and plays a crucial role in learning, cognition, attention, emotional activity, and memory formation [3]. About 60% of neurons in the brain, including all cortical pyramidal neurons and thalamic relay neurons, use glutamate as their primary neurotransmitter. Dysfunction of the neurotransmitter glutamate plays a significant role in the pathophysiology of epilepsy. The release of glutamate leads to depolarization of the postsynaptic neuron due to interaction with inotropic AMPA and NDMA receptors. This continues until glutamate is captured and converted into glutamine, which does not produce the described effect. When glutamate elimination mechanisms are disrupted, its concentration increases in the synaptic clefts of certain (epileptogenic) areas of the brain, leading to excitotoxicity, damage, and death of nerve cells. Elevated glutamate concentrations was determined in the hippocampus of patients suffering from epilepsy before and during attack. Neurotoxic The effect of glutamate increases the transmembrane flow of Ca^{2+}, in turn, the excess level of which can contribute to the uncontrolled activation of neurons during an epileptic seizure [5,6]. It has been found that increased amounts of glutamate have a neurotoxic effect, causing neuronal apoptosis, as well as neuropsychiatric disorders, including epilepsy.

The concentration of glutamate in blood serum samples was determined spectrophotometrically using Sigma - aldrich reagent kits (Glutamate Assay Kit) on a spectrofluorimeter Solar CM2203 (Solar, Belarus) at 450 nm. This glutamate assay is based on glutamate dehydrogenase-catalyzed oxidation of glutamate, resulting in the formation of nicotinamide adenine dinucleotide, which reduces the formazan (MTT) reagent. The color intensity of the product, measured at 565 nm, is proportional to the glutamate concentration in the sample.

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The aim of the study was to determine changes in serum glutamate concentrations in patients with focal epilepsy.

2. Material and Methods

Twenty-six serum samples from patients diagnosed with focal epilepsy were analyzed. The study included men and women aged 18–50 years with newly diagnosed or previously verified symptomatic epilepsy. Exclusion criteria included severe neurological deficits (aphasic and movement disorders), the presence of an active neurological disease, and severe concomitant somatic pathology. The observation period was 3 months. The initial examination included collection of anamnestic data, clinical and neurological examination, laboratory blood tests (general and biochemical analyses), ECG recording, computed tomography or magnetic resonance imaging of the brain to exclude an acute or ongoing neurological process and clarify the type of epilepsy, as well as an electroencephalographic study.

The concentration of glutamate in blood serum was determined by spectrophotometry. The Glutamate Determination Kit "Glutamat" The Assay Kit is highly sensitive and allows for the quantitative assessment of glutamate levels in various biological samples. Serum glutamic acid concentrations were expressed in nmol /ml. Laboratory testing was performed upon admission to the clinic before medication was prescribed. Blood for analysis was collected in the morning on an empty stomach. To separate serum from formed elements, the blood tubes were centrifuged.

The study included three visits: Visit 1—before the start of therapy, Visit 2—at the 4th week of treatment, and Visit 3—at the 12th week of therapy. All patients were prescribed monotherapy. Antiepileptic drugs: carbamazepine at a daily dose of 600 mg or zonisamide at a daily dose of 300 mg. Depending on the therapy administered, patients were divided into two groups: Group I included patients receiving carbamazepine, and Group II included patients receiving zonisamide as an antiepileptic drug.

Statistical processing of the research results was performed on a personal computer using the STATISTICA 8.0 software package.

3. Results of the Study

The study included 26 patients with a verified diagnosis of focal epilepsy, including 17 men and 9 women. The average age of men was 38.59 ± 8.14 years, women — 36.21 ± 5.06 years. In the clinical picture of the disease, secondarily generalized epileptic seizures predominated, which were registered in 69.22% (n = 18) of patients. Complex focal seizures with automatisms were noted in 23.08% (n = 6) of patients, simple focal seizures — in 7.7% (n = 2). Focal seizures without impaired consciousness were observed in 19 (73.1%) patients, focal seizures with impaired consciousness — in 7 (26.9%). According to the frequency of epileptic seizures, patients were distributed as follows: seizures less than once a month were noted in 9 (34.6%) patients; Several times a month, but less than once a week — in 12 (46.15%); several times a week, but less than once a day — in 3 (11.55%); once a day or more — in 2 (7.7%) patients. All patients received antiepileptic therapy as monotherapy. Carbamazepine was used in 57.7% (n = 15) of patients, zonisamide — in 42.3% (n = 11). The dosage of carbamazepine was carried out from 200 mg per day with an increase of 100 mg to 400 mg per day in two doses. Titration of the zonisamide dose was mandatory: in adults - from 50 mg per day, increasing by 50 mg once every 1-2 weeks. Evaluation of the effectiveness of antiepileptic therapy was carried out on the basis of the dynamics of the frequency of epileptic seizures and treatment tolerability. A complete absence of attacks (100%) was regarded as drug-induced remission; therapy was considered effective when the frequency of attacks was reduced by 50% or more, and ineffective when the frequency of attacks was reduced by less than 50%.

The analysis of serum glutamate levels revealed that glutamate concentrations in all studied patient groups were statistically significantly higher than those in healthy individuals (19.2 ± 8.5 nmol /ml). The lowest glutamate levels among all examined patient groups were found in patients with focal seizures without impairment of consciousness (15.2 ± 3.5 nmol /ml). The highest serum amino acid levels were found in patients with secondarily generalized epileptic seizures (21.7 ± 10.64 nmol /ml). However, significant differences were found between the glutamate levels in patients with focal seizures without impairment and those in patients with secondarily generalized epileptic seizures, who had the highest serum amino acid levels ($p = 0.055$).

A study of serum glutamate levels before and after therapy revealed no significant differences in the groups of patients taking carbamazepine as an antiepileptic therapy. However, significant differences in glutamate levels after therapy were found in the group of patients taking zonisamide.

4. Conclusions

Determination of glutamate concentration in blood serum, a biological material available for diagnostic research, using a technique that is easily reproducible in most clinical laboratories, may become one of the new methods for the differential diagnosis of epilepsy.

The differences in the level of glutamate in the peripheral blood of patients with focal epilepsy after a course of treatment with zonisamide indicate a beneficial effect of this drug on glutamatergic neurotransmission.

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