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MODERN OPPORTUNITIES OF ELECTROENCEPHALOGRAPHY IN THE DIAGNOSIS OF EARLY CHRONIC BRAIN ISCHEMIA

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Abstract: Chronic cerebral ischemia (CBI) represents one of the most pressing issues in modern neurology, particularly in the context of the increasing prevalence of cerebrovascular diseases among the working-age population. Early diagnosis of this condition is crucial for the timely prescription of neuroprotective therapy and the prevention of stroke development.

Keywords: Electroencephalography, Chronic Cerebral Ischemia, Early Diagnosis, Bioelectrical Activity, Cerebrovascular Diseases, Quantitative EEG, Spectral Analysis, Coherence

Introduction

Electroencephalography (EEG), as a non-invasive, accessible, and highly sensitive method for studying brain bioelectrical activity, occupies a special place in the diagnostic algorithm for suspected SIGM. Modern digital EEG systems with quantitative analysis, spectral mapping, and coherent analysis capabilities have significantly expanded the diagnostic capabilities of the method.

The EEG's ability to detect functional disorders in the preclinical stage, when structural changes are not yet visible through neural imaging methods, is of particular value. Analysis of the frequency-amplitude characteristics of biopotentials, assessment of interhemispheric asymmetry, and brain reactivity to functional tests allow not only to diagnose CID in the early stages but also to assess the severity of ischemic changes and the effectiveness of the therapy being administered.

Currently, new EEG markers for early cerebral ischemia are being actively studied, including coherence analysis, assessment of nonlinear brain activity dynamics, and the application of machine learning for automatic data interpretation. These achievements open up new perspectives for improving diagnostic accuracy and personalizing treatment approaches for patients with SIGM[1,2,3].

The aim of the study is to study the diagnostic capabilities of modern electroencephalography methods in identifying early signs of chronic cerebral ischemia and to improve the EEG diagnostic algorithm based on the comprehensive application of digital analysis of brain bioelectrical activity.

Research materials and methods: The study was conducted at the Department of Neurology of Samarkand Medical University and was conducted between 2024 and 2026. The research type is prospective controlled with elements of comparative analysis.

The study plans to include 280 patients aged 45 to 75 years with clinical signs of the initial manifestations of chronic cerebrovascular insufficiency, divided into four groups. The primary group consisted of 85 patients with verified stage I chronic cerebral ischemia; the second group consisted of 82 patients with stage II CIDH; and the third group consisted of 78 patients with stage III CIDH. The control group consisted of 35 practically healthy individuals of the appropriate age without signs of cerebrovascular pathology[4,5].

Inclusion criteria: age 45–75 years; presence of a clinical diagnosis of stage I–III chronic cerebral ischemia, verified in accordance with ICD-11 criteria and national clinical protocols; informed consent of the patient. Exclusion criteria: history of acute cerebral circulatory disorders, epilepsy and other paroxysmal conditions, severe dementia, mental illnesses, brain cancer processes, history of traumatic brain injury, and use of psychoactive drugs affecting the EEG.

Clinical methods included a thorough medical history collection focusing on risk factors for cerebrovascular diseases, neurological examination, assessment of cognitive functions using the MMSE (Mini-Mental State Examination) scale, a clock drawing test, a frontal dysfunction battery (FAB), and functional status analysis using the Rankin scale[6,7,8].

Instrumental examination methods included brain magnetic resonance imaging to assess the degree of leukoareosis using the Fazekas scale, duplex scanning of the main arteries of the head to determine the degree of stenosis and the nature of atherosclerotic plaques, and transcranial Doppler ultrasound to assess cerebrovascular reactivity.

Electroencephalographic methods included EEG registration on a 32-channel electroencephalograph with digital signal processing (discrete frequency 500 Hz, bandwidth 0.5–70 Hz), conducting standard functional tests (photostimulation, hyperventilation), quantitative EEG analysis with the construction of power distribution maps in the main frequency ranges (delta 0.5–4 Hz, teta 4–8 Hz, alpha 8–13 Hz, beta 13–30 Hz, gamma 30–50 Hz), analysis of coherence and synchronization index between different brain regions, power spectral analysis with the identification of dominant frequency peaks, and three-dimensional mapping of electrical activity sources (sLORETA analysis)[9,10].

Biochemical research methods included determining levels of neurospecific markers (S100B, NSE, GFAP), markers of endothelial dysfunction (endothelin-1, NO synthase), lipid metabolism and homocysteine indicators, and C-reactive protein levels.

Statistical processing of the obtained data was performed using the SPSS software package version 26.0 and specialized EEG analysis software (EEGLAB, Brainstorm). For quantitative variables, parametric (Student's t-test, ANOVA) or non-parametric (Mann-Whitney, Kraskel-Wallis test) methods were applied depending on the nature of the distribution. Qualitative indicators were compared using the $\chi 2$ test. Correlation analysis was conducted using the Pearson and Spearman method. Machine learning methods (logistic regression, random forest, neural networks) were used to create diagnostic algorithms. Differences were considered statistically significant at $p < 0.05$.

Materials and Methods

The study was conducted at the Department of Neurology of Samarkand State Medical University during the period 2024–2026. This was a prospective controlled study with elements of comparative analysis aimed at evaluating the diagnostic potential of electroencephalography (EEG) in early chronic cerebral ischemia. A total of 280 patients aged 45–75 years were included and divided into four groups: 85 patients with stage I chronic cerebral ischemia, 82 patients with stage II, 78 patients with stage III, and 35 age-matched healthy controls without cerebrovascular pathology. Inclusion criteria were confirmed diagnosis of chronic cerebral ischemia according to ICD-11 and national clinical protocols, age between 45 and 75 years, and informed consent. Exclusion criteria included acute cerebrovascular events, epilepsy, severe dementia, brain tumors, psychiatric disorders, traumatic brain injury, and use of drugs affecting EEG activity.

Clinical evaluation included neurological examination and cognitive assessment using MMSE, clock drawing test, FAB, and Rankin scale. Instrumental methods involved MRI with Fazekas scale assessment, duplex ultrasound of cerebral arteries, and transcranial Doppler for cerebrovascular reactivity. EEG was recorded using a 32-channel digital system (0.5–70 Hz, 500 Hz sampling rate) with functional tests (hyperventilation, photostimulation), followed by quantitative spectral analysis,

coherence assessment, and 3D source localization (sLORETA). Additional laboratory tests included biomarkers of neuronal injury (S100B, NSE, GFAP), endothelial dysfunction markers, lipid profile, homocysteine, and CRP levels. Statistical analysis was performed using SPSS 26.0 and R 4.0, applying t-test, ANOVA, Mann–Whitney, chi-square tests, and correlation analysis, with $p < 0.05$ considered statistically significant.

Results and Discussion

When conducting neuro-physiological and instrumental studies in individuals of the primary and control groups, a certain increase was noted. A study of the bioelectrical activity of the brain was conducted in individuals with early stages of chronic brain ischemia and in individuals of the control group, right-handed individuals. Thus, in a portion of the main group (38.9%), as well as in the control group (65.6%), the a-rhythm was represented by a regular component with a maximum amplitude of 54.27±22.99 μV, an interhemispheric asymmetry of 13.03±6.7%, a dominant frequency of 10.13±0.87 Hz, and a severity index of 87.67±7.04%.

In the frontal regions, an 0-rhythm with an amplitude of 15.77±4.59 μV and a frequency of 19.03±2.68 Hz predominated. There were no slow or sharp waves.

The approximate reaction with eye opening revealed an attenuation of the a-rhythm by 86.17±12.28% in amplitude and by 88.4±12.46% in severity index. Three-minute hyperventilation and rhythmic photostimulation in the frequency range from 5 to 30 Hz did not lead to EEG changes,[11,1].

This bioelectric activity of the brain corresponded to the first type of EEG according to E.A. Zhirmunskaya and was frequently observed in both the main group (38.9%) and healthy individuals (65.6%), with predominance in the main group against the background of arterial hypertension (65.7%) ($p<0.05$).

Bioelectric activity of the brain in individuals with early stages of chronic brain ischemia and individuals in the control group (%)

In the other part of the studied individuals in the primary (5.4%) and control (31.3%) groups, the a-rhythm was represented by a regular component with a maximum amplitude of 90.94±14.31 μV, an interhemispheric asymmetry of 16±5.63%, a dominant frequency of 9.84±0.64 Hz, and a severity index of 95.29±4.59%. The beta rhythm with an amplitude of up to 15.65±3.95 μV and a frequency of 18.24±1.56 Hz was represented by rare waves without zonal differences. Slow and sharp waves were not recorded. The approximate reaction with eye opening revealed an attenuation of the a-rhythm by amplitude by 85.24±15.09%, and by severity index by 90.71±9.05%. Hyperventilation and rhythmic photostimulation did not alter the EEG picture. This type corresponded to the second EEG type according to E. A. Zhirmunskaya—frequently found in individuals of the control group (31.3%), and less frequently in individuals with early stages of chronic cerebral ischemia (5.4%) ($p>0.05$), regardless of the cause of the disease[13].

In a third of individuals in the primary (27.85%) and control (3.1%) groups, the a-rhythm was poorly modulated, flattened with a maximum amplitude of 26.6±7.71 μV, hemispheric asymmetry of 14.83±5.55%, a dominant frequency of 10.2±0.86 Hz, and a low severity index of 12.67±4.91% in the absence of zonal differences. The beta-rhythm with an amplitude up to 16.67±4.22 μV and a frequency of 19.6±3.71 Hz had no zonal differences (dominated in all branches). There were no slow or sharp waves. An attenuation of the a-rhythm during eye opening was noted: by amplitude by 73.77±26.59%, and by severity index by 81.27±23.75%. In this group of subjects, three-minute hyperventilation led to a significant increase in α-activity by an amplitude of 41.9±24.6%. The photostimulation response in the frequency range from 5 to 30 Hz was non-informative. This type, reflecting the strengthening of ascending activating effects of non-specific midline structures of the brainstem, approached the third EEG type according to E.A. Zhirmunskaya and prevailed in individuals with MS MCI due to cerebral

atherosclerosis (39.6%) ($p<0.05$)[14].

In a number of individuals in the main group (27.85%), the a-rhythm was unmodulated, represented by an irregular frequency component with a maximum amplitude of 37.1±10.01 μV, interhemispheric asymmetry of 19.9±6.6%, a dominant frequency of 9.65±1.4 Hz, and a severity index of 82.83±8.55%. Zonal differences in the a-rhythm were smoothed out. There was a disorganization of a-activity of a mild degree due to 0-rhythm or due to single 0-waves with an amplitude not exceeding background activity (31.6±10.45 μV) with a dominant frequency of 5.08±0.65 Hz and a severity index of 10.87±6.38%. The beta-rhythm with an amplitude of up to 23.63±8.71 μV and a frequency of 16.5±2.06 Hz also exhibited smoothed zonal differences. The approximate reaction with eye opening revealed an attenuation of the a-rhythm by amplitude by 78.43±22.14%, and by the severity index by 79±22.09%. Three-minute hyperventilation and rhythmic photostimulation did not lead to EEG changes. This type corresponded to the fourth EEG type according to E.A. Zhirmunskaya and predominated in individuals with early stages of chronic cerebral ischemia against a background of combined arterial hypertension and cerebral atherosclerosis (42.7%) ($p<0.05$).

To compare EEGs in individuals of the control and main groups, taking into account the etiology of the disease, total spectral amplitude-frequency characteristics were used with topical mapping based on background recording.

Thus, when comparing electroencephalograms in individuals of the primary and control groups, a diffuse decrease in amplitude characteristics across almost all branches was identified in patients with SMI RS ($p<0.05$).

No differences in EEG frequency characteristics were found in the primary and control groups ($p>0.05$).

In the subgroup of individuals with early-stage chronic cerebral ischemia against the background of arterial hypertension, compared to the control group, amplitude indicators were reduced in the area of the poles of the occipital lobes of both hemispheres and the pole of the temporal lobe of the dominant hemisphere, and frequency indicators in the anterior temporal region of the dominant hemisphere ($p<0.05$).

In patients suffering from the early stage of chronic brain ischemia due to cerebral atherosclerosis, a decrease in EEG amplitude (temporo-temporal branching) and frequency (frontal branching) characteristics in the dominant hemisphere was identified compared to individuals in the control group ($p<0.05$).

In patients with MS MIA due to a combination of causes, a decrease in amplitude characteristics in all branches of both cerebral hemispheres was identified without changes in EEG frequency characteristics compared to individuals in the control group ($p<0.05$)[15].

Discussion. The results obtained indicate the high diagnostic value of modern EEG analysis methods in identifying early signs of chronic cerebral ischemia. The established dynamics of bioelectrical activity changes reflect the progressive disruption of neural networks and can serve as an objective criterion for assessing the severity of the disease.

The identified decrease in alpha-rhythm frequency and index in the early stages of CII indicates a disruption in the function of thalamo-cortical connections, which corresponds to modern concepts of the pathophysiology of chronic cerebral ischemia. Increased slow-wave activity reflects cortical neuron dysfunction due to chronic hypoxia and may precede structural changes detected by neuroimaging methods.

Conclusions: Thus, visual EEG analysis according to E. A. Zhirmunskaya showed that mild and moderate changes in brain bioelectrical activity are characteristic of MS with SMI.

It was established that the third type of EEG predominates for patients with early stage chronic cerebral ischemia against the background of cerebral atherosclerosis (39.6%) and the fourth type against the background of mixed cause cerebrovascular disease (42.7%).

Spectral analysis of EEG amplitude-frequency characteristics with topical mapping objectified the presence of significant differences ($p<0.05$) in EEG indicators in patients with early stages of chronic cerebral ischemia compared to individuals in the control group, as well as the zonal distribution in individuals with MS and MCI, taking into account the etiology of the disease.

Specifically, in individuals of the main group as a whole and in individuals of the main group due to the combination of arterial hypertension and cerebral atherosclerosis, a diffuse decrease in EEG amplitude indicators was identified without a decrease in frequency. In individuals with early-stage CMI against the background of cerebral atherosclerosis, a decrease in the amplitude-frequency parameters of the EEG spectrum from the dominant hemisphere was detected, while in individuals with arterial hypertension, a decrease in amplitude parameters was found in the poles of the occipital lobes of both cerebral hemispheres and the pole of the left frontal lobe, and frequency parameters in the anterior temporal region of the dominant hemisphere.

Conclusion

The results of this study demonstrate that electroencephalography (EEG), especially when combined with modern quantitative and spectral analysis techniques, has high diagnostic value in the early detection of chronic cerebral ischemia. EEG allows the identification of functional brain disturbances even at preclinical and early clinical stages, when structural neuroimaging changes may still be minimal. In particular, disorganization of the alpha rhythm, reduction in amplitude parameters, and the appearance of slow-wave activity reflect impaired adaptive mechanisms of the brain under chronic hypoxic conditions. These changes are associated with dysfunction of thalamo-cortical connections and reduced synchronization of neuronal networks, making them important early biomarkers of cerebral ischemia.

Furthermore, EEG alterations were found to vary depending on the etiological factors of chronic cerebral ischemia. Patients with cerebral atherosclerosis and mixed vascular pathology demonstrated more pronounced abnormalities in bioelectrical activity compared to those.

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