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CLINICAL FEATURES AND TREATMENT EFFICACY OF POST-TRAUMATIC EPILEPSY AND COGNITIVE IMPAIRMENT IN CHILDREN WITH TRAUMATIC BRAIN INJURY

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ABSTRACT

Objective: To investigate the clinical features of post-traumatic epilepsy (PTE) and cognitive impairment in children following traumatic brain injury (TBI), and to assess the therapeutic efficacy of levetiracetam and pantogam.

Methods: Thirty children diagnosed with TBI were divided into three clinical groups: Group I — PTE only (n=16), Group II — cognitive impairment only (n=2), and Group III — combined PTE and cognitive impairment (n=12). All patients underwent EEG (19-channel, 10–20 system), MSCT (within 24 hours), and neuropsychological assessment using MoCA and Raven's Progressive Matrices. Treatment duration was 6 months. Statistical analysis included paired t-test and Fisher's exact test ($p<0.05$).

Results: Epileptiform activity was detected in 87.5% of Group I and 91.7% of Group III patients on EEG. The most common MSCT findings were contusion foci (60.0%) and focal atrophy (60.0%). Levetiracetam reduced seizure frequency by 54.8%; combined therapy in Group III achieved a 76.5% reduction. MoCA scores improved significantly in all groups (Group I: $\Delta=+4.4$; Group III: $\Delta=+5.6$; $p<0.001$).

Conclusion: Combined PTE and cognitive impairment following TBI worsens clinical prognosis. The combination of levetiracetam and pantogam demonstrated efficacy even in the most severe cases, with a synergistic effect on both seizure control and cognitive recovery.

Keywords: traumatic brain injury, post-traumatic epilepsy, cognitive impairment, levetiracetam, pantogam, EEG, pediatric neurology.

INTRODUCTION

Traumatic brain injury (TBI) is an acquired brain injury resulting from an external mechanical force, which may cause temporary or permanent functional impairment. TBI is also referred to as 'acquired brain injury,' 'head injury,' or 'brain damage,' and results in disruption of normal brain function due to sudden trauma. TBI may lead to physical, psychological, cognitive, emotional, and social consequences [1].

Traumatic brain injury (TBI) affects approximately 69 million people annually [2] and is associated with numerous chronic somatic and cognitive disorders, among which epilepsy is particularly significant [3,4]. Post-traumatic epilepsy (PTE) is a long-term condition following severe or moderate TBI, characterized by recurrent unprovoked seizures occurring 7 or more days after the injury [5,6]. The development of PTE represents an additional clinical burden for patients and is associated with profound neurological changes, causing cognitive dysfunction and increased mortality [7,8].

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Aim of the study to investigate the clinical features of disease through neuropsychological and paraclinical examinations in 30 children with traumatic brain injury, and to improve treatment using levetiracetam and pantogam.

Study objectives

1. To conduct a comprehensive clinical-neurological examination of children with traumatic brain injury.
2. To identify age-related changes in children with traumatic brain injury through cognitive and neuropsychological testing.
3. To perform neurovisualization (MSCT, EEG) examinations in children with traumatic brain injury and to improve treatment using pantogam and levetiracetam.

Materials and methods

This study included 30 children diagnosed with traumatic brain injury (TBI) in whom paroxysmal conditions and cognitive impairments were identified during the follow-up period. Patients were divided into three clinical groups: Group I — children with post-traumatic epilepsy only (n=16); Group II — children with cognitive impairment only (n=2); Group III — children with both pathologies simultaneously (n=12). Cognitive function was measured using two validated instruments. The Montreal Cognitive Assessment (MoCA) adapted for children assessed attention, memory, executive functions, language, and visuospatial abilities on a 30-point scale. Raven's Coloured Progressive Matrices were used to evaluate logical reasoning and executive functions. Both tests were administered before and after treatment to assess cognitive dynamics.

Electroencephalography (EEG) was performed in all participants using standard 10–20 electrode placement during wakefulness and, where possible, during natural sleep. Multi-slice computed tomography (MSCT) was performed within 24 hours of admission; repeat imaging was performed when clinically indicated. EEG findings were classified as normal, focally abnormal (focal slow waves or epileptiform discharges), or diffusely abnormal (generalized slowing).

Data were processed using Excel. Categorical variables were presented as frequencies and percentages. Continuous variables were expressed as mean and standard deviation. Pre- and post-treatment results were compared using paired t-test. Inter-group comparisons were performed by age group and TBI severity. Fisher's exact test was used for categorical associations. Statistical significance was set at $p<0.05$.

Clinical-Neurological Examination Findings

During clinical examination, the following neurological signs were recorded to varying degrees in all three groups: nystagmus, changes in muscle tone (hypotonus and hypertonus), memory and logical reasoning impairments, speech disorders, reduced deep reflexes, hemiparesis, and monoparesis. These signs were observed with the highest frequency and severity in Group III patients, while symptoms were mild or absent in Group I.

Cognitive Features by Age Group

Neuropsychological and cognitive testing of children with TBI revealed that age-related differences were clearly evident not only in clinical presentation but also in the dynamics of

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recovery. In young children (ages 3–6), although neural plasticity is high, the risk of delayed cognitive deficits — particularly in attention, memory, and executive functions — is significantly increased. In middle-aged children (ages 7–10), speech and verbal memory impairments predominate, as hemispheric dominance has not yet been fully established. In older children (ages 10–14), cerebellar symptoms and executive function deficits are more pronounced and persist longer.

MSCT Findings

All patients were examined by multi-slice computed tomography (MSCT) at baseline. The most common MSCT findings across the overall group were cerebral contusion foci (n=18; 60.0%). Post-traumatic focal atrophy was found in 14 patients (46.7%), moderate or severe ventriculomegaly in 8 patients (26.7%), and subdural hematomas in 5 patients (16.7%). Four patients (13.3%) — all from Group I — showed no significant changes on MSCT.

Table 1. MSCT Findings by Group

MSCT Findings	Group I	Group II	Group III	Total
Contusion foci	10 (62.5%)	1 (50.0%)	7 (58.3%)	18 (60.0%)
Focal atrophy	9 (56.3%)	1 (50.0%)	8 (66.7%)	18 (60.0%)
Ventriculomegaly	3 (18.8%)	1 (50.0%)	4 (33.3%)	8 (26.7%)
Subdural changes	2 (12.5%)	0 (0%)	3 (25.0%)	5 (16.7%)
Diffuse axonal injury	2 (12.5%)	1 (50.0%)	5 (41.7%)	8 (26.7%)
No changes detected (%)	4 (25.0%)	0 (0%)	0 (0%)	4 (13.3%)

Electroencephalography Findings

All patients were examined with a 19-channel EEG recording using the standard 10–20 system. Recordings were made at rest and during functional tests (hyperventilation, photostimulation).

In Group I (epilepsy only, n=16), epileptiform activity was detected in 14 patients (87.5%) on EEG. Of these, 9 (56.3%) had focal epileptiform discharges — predominantly in the frontal

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and temporal regions. Five (31.2%) showed generalized epileptiform changes. The remaining 2 (12.5%) had normal EEG, with the diagnosis confirmed based on the clinical history of epileptic seizures.

In Group II (cognitive impairment only, n=2), no epileptiform changes were detected. Both patients showed background slowing — diffuse theta-delta waves — characteristic of diffuse cortico-subcortical dysfunction.

In Group III (epilepsy + cognitive impairment, n=12), EEG changes were the most numerous and severe. Epileptiform activity was detected in 11 patients (91.7%). Of these, 7 (58.3%) had focal and 4 (33.3%) had generalized discharges. Simultaneously, background rhythm slowing was recorded in all 12 patients (100%).

Table 2. EEG Findings by Group

EEG Findings	Group I	Group II	Group III
Epileptiform activity (%)	14 (87.5%)	0 (0%)	11 (91.7%)
Focal epileptiform discharges (%)	9 (56.3%)	0 (0%)	7 (58.3%)
Generalized discharges (%)	5 (31.2%)	0 (0%)	4 (33.3%)
Background rhythm slowing (%)	7 (43.8%)	2 (100%)	12 (100%)
Normal EEG, n (%)	2 (12.5%)	0 (0%)	0 (0%)

Treatment Outcomes

In Group I patients, seizure frequency decreased significantly following levetiracetam administration. The mean monthly seizure count was 4.2±1.8 at baseline and fell to 1.9±1.0 after treatment, representing a 54.8% reduction ($p<0.001$). Complete remission was achieved in 6 of 16 patients (37.5%). In the remaining 10 (62.5%), seizure frequency decreased by at least 50%.

In Group II patients, pantogam administration over 6 months provided stable improvement in cognitive functions. No adverse effects were observed. In Group III, combined pharmacotherapy showed the most notable positive dynamics. Seizure frequency fell from 5.1±2.2 to 1.2±0.9 (76.5% reduction; $p<0.001$). Complete remission was achieved in 3 of 12 patients (25.0%).

Post-Treatment Cognitive Dynamics: MoCA and Raven Test Results

After completion of the complex treatment course (mean follow-up period 6 months), all patients underwent repeat cognitive assessment. Post-treatment MoCA results showed score improvement in all three groups (Table 3). A similar positive trend was also observed on the Raven's Progressive Matrices test (Table 4).

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Table 3. MoCA Test Results (Before and After Treatment)

Group	Before treatment	After treatment	Δ (difference)	p-value
Group I (16)	19.4±3.1	23.8±2.6	+4.4	<0.01
Group II (2)	17.0±2.8	21.5±2.4	+4.5	<0.05
Group III (12)	13.6±4.2	19.2±3.8	+5.6	<0.001

Table 4. Raven Test Results (Before and After Treatment)

Group	Before treatment	After treatment	p-value
Group I (16)	22.4±5.6	27.1±4.8	<0.01
Group II (2)	19.5±4.3	24.0±3.9	<0.05
Group III (12)	15.8±5.9	21.6±5.1	<0.01

DISCUSSION

This study confirmed that the coexistence of PTE and cognitive impairment in children following TBI significantly increases clinical severity. According to literature data, the incidence of PTE after severe TBI ranges from 10–25% [5,8]; however, in our study, this figure reached 53.3% for the overall group, which may reflect selection bias toward hospitalized patients with higher severity characteristics.

Our EEG findings are consistent with those of Englander et al. (2003): frontal-temporal localization of focal epileptiform discharges is considered the primary mechanism of epileptogenesis in TBI [6]. The fact that background alpha rhythm slowing covered 100% of Group III patients was assessed as clinical-EEG confirmation of diffuse cortico-subcortical damage, which, as described by Bramlett and Dietrich (2015), may be associated with long-term neurodegenerative processes [3].

The predominance of multifocal and diffuse lesions in Group III on MSCT (83.3%) explains the pathogenetic mechanisms of concurrent PTE and cognitive deficit. The correlation between lesion volume and MoCA scores (unifocal: 20.1±3.4; multifocal: 14.3±4.1; $p<0.05$) confirms the diagnostic value of neuroimaging in predicting cognitive prognosis [2,7].

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The antiepileptic efficacy of levetiracetam (54.8% reduction in Group I, 76.5% in Group III) is consistent with ILAE recommendations and available clinical trial data [4,5]. The synergistic effect observed with the addition of pantogam is explained by parallel modulation of the GABAergic system by both drugs at different receptor levels. This combination warrants broader clinical trials in pediatric neurology.

Study limitations: The small sample size of Group II (n=2) makes it difficult to draw statistical conclusions for this group. The absence of a control group does not allow comparison of drug efficacy with placebo. The follow-up period (6 months) is insufficient for assessing long-term prognosis. Future prospective multicenter studies with control groups are recommended.

CONCLUSIONS

1. During clinical examination, neurological signs were recorded to varying degrees in all three groups. These signs were observed with the highest frequency and severity in Group III patients.

2. Cognitive and neuropsychological testing of children with TBI confirmed age-related differences: younger children have a higher risk of delayed cognitive deficits; middle-aged children predominantly show speech and verbal memory impairments; older children show more pronounced and prolonged cerebellar symptoms and executive function deficits.

3. EEG examination confirmed a high frequency of epileptiform activity in Groups I and III (87.5% and 91.7%, respectively). MSCT findings revealed predominant frontal and temporal lesions; multifocal and diffuse lesions were significantly more common in Group III and directly correlated with clinical severity and cognitive indicators.

4. The combination of levetiracetam and pantogam is indicated for the coexistence of epilepsy and cognitive impairment. It is recommended that treatment duration in this group be no less than 12 months, and a MoCA score of 26 or higher should be accepted as a clinical criterion of full treatment efficacy.

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