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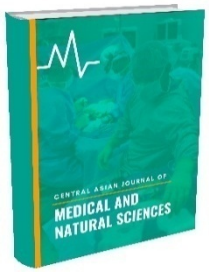

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Diagnostic Dynamics of Post-Traumatic Epilepsy in the Context of Comorbid Medical Conditions

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ABSTRACT: Posttraumatic epilepsy is a form of epilepsy in which the formation of an epileptogenic and epileptic focus has a reliable causal relationship with previously suffered traumatic brain injury and associated CVD, which may clinically manifest as recurrent unprovoked epileptic seizures. This suggests that it is reasonable to identify patients with this form of the disease as a separate study group [1,3,5].

Keyword: epilepsy, seizures, epileptic status, trauma, EEG, paroxysmal index, MRI.

Introduction: Post-traumatic epilepsy (PTE) can be characterized as one of the variants of the consequences of traumatic brain injury (CBI) with a leading epileptic syndrome, which is manifested, respectively, by systematically recurrent epileptic seizures, most often of convulsive nature. The leading aetiological factor in symptomatic epilepsy in young adults is traumatic brain injury, which accounts for 30-50% of all peacetime injuries. [5,6]. The incidence of post-traumatic epilepsy in cases of previous traumatic brain injury ranges from 5 to 50%, according to numerous studies, and its course is often progenerative. The diagnosis and drug treatment of post-traumatic epilepsy remains one of the most challenging tasks in clinical neurology. In spite of advances in modern neurology, many questions remain, in particular, the problem in the course of the disease in people with PTE who have concomitant cardiovascular disease has been insufficiently studied. Introduction. Posttraumatic epilepsy (PTE) can be characterized as a variant of traumatic brain injury (CBI) with a leading epilepsy syndrome manifested by systematic recurrent epileptic seizures, most often of convulsive nature. The leading aetiological factor in symptomatic epilepsy in young adults is traumatic brain injury, which accounts for 30-50% of all peacetime injuries[11]. The incidence of post-traumatic epilepsy in cases of previous traumatic brain injury ranges from 5 to 50%, according to numerous studies, and its course is often progenerative. The diagnosis and drug treatment of post-traumatic epilepsy remains one of the most challenging tasks in clinical neurology. Despite the advances of modern neurology, many questions remain, in particular the problem in people with PTE who suffer from concomitant cardiovascular disease. Age-related changes in the brain reduce cerebrovascular reserve and increase its sensitivity to ischemia [2,9,10]. The clinical manifestations of a seizure correspond to an area of circulatory insufficiency. According to researchers], precursory seizures (seizures preceding the development of stroke) are most often partial and may indicate the localization of an ischemic focus [4, 8,12].

Purpose of the study: To study the dynamics of clinical and neurophysiological changes against the background of complex medical therapy in PTE patients with CVD.

Material and Methods: We examined 110 patients with PTE who were under observation in the Department of Neurology 1 of the SamMI clinic. The patients ranged in age from 40 to 65 years. The duration of disease ranged from 6 to 10 years, men 74%, women 26%, as well as 48 patients in whom PTE was not detected (comparison group).

The study group was divided into 3 groups according to the relevant criteria, this is presented in Table 1.

Table 1.

Main group	Inclusion criteria	number of patients
I subgroup	PTE + PRTRS	65
II subgroup	PTE	45
Control group	Epilepsy	48

The main group consisted of 110 patients with post-traumatic epilepsy in whom the diagnosis was established or confirmed on the basis of having already had repeated (at least two) unprovoked epileptic seizures. The control group consisted of 48 patients with epilepsy who were not diagnosed with post-traumatic epilepsy during follow-up.

All patients underwent a comprehensive examination, including neurological status, EEG, MRI (MSCT), transcranial dopplerography of cerebral vessels and neuropsychological examination, laboratory methods (blood biochemistry), consultation with narrow specialists. The diagnosis of forms of epilepsy and types of epileptic seizures was established according to the International Classification of Diseases.

Results and discussion: In the majority of patients with posttraumatic epilepsy we examined, seizures were partial with secondary generalization (60.0%), and the clinical picture of the partial component predominantly corresponded to temporal mediabasal localization of the epileptic focus (temporal pseudoabsences, autovisceral, automorphisms). In addition, a significant proportion of patients had only generalised seizures without a partial component (29.3%) or polymorphic seizures (16.0%). As for the frequency of seizures, in most cases it was high - more than 3 times a year to once a month and more than once a month (30.7% and 20.0% cases, respectively). All patients underwent brachiocephalic artery (BCA) CTDG during the interictal period (at least 3 days after the attack). Velocity and resistive indices were analyzed, features of cerebrovascular reserve were studied using functional tests. The obtained indices of cerebral blood flow in patients with different forms of PTE were compared with each other, with indices in PTE patients with hemodynamically significant changes of BCA and with control indices.

The examination of PTE patients revealed that the level of cerebrovascular reactivity and decreased blood flow velocity in the intracranial segments of VAS on the side of the lesion were the most informative parameters, valuable in prognostic terms. This was evidenced by the significant difference in the analyzed parameters between the groups.

Table 2

Cerebrovascular reactivity and blood flow rate in PTE

	Number of Patients	LSC	PI	R	LSC	PI	R
Group 1(PTE patients with CVD)	65	65,3± 3,2	0,77± 0,03	1,21± 0,025	40± 0,5	0,44± 0,03	1,04± 0,01
Group 2(PTE patients)	45	56± 5,6	0,72± 0,08	1,27± 0,061	31± 1,4	0,54± 0,11	1,09± 0,02
3 group(control	48	70± 4,9	0,76±	1,3± 0,03	38± 2,8	0,58±	1,15±

group)			0,03			0,03	0,03

Cerebrovascular reactivity serves as a quantitative characteristic of the cerebral circulation regulation system and reflects the state of perfusion reserves. In group 1 of patients with PTE and CVD, cerebrovascular reactivity in the carotid basins was assessed in 57 patients and in the vertebrobasilar basin in 52 patients.

In Group 2, the state of perfusion reserves in the carotid system was studied in 38 patients, and in the vertebro-basilar basin in 37 patients. All groups of patients studied had normal or reduced (less than 25%) cerebrovascular reactivity. No individuals with increased cerebrovascular reactivity were detected.

No significant differences in decreased perfusion reserves in carotid pools were found in patients with different pathogenetic subtypes of stroke ($\chi2=2.64$; $p=0.44$): in the atherothrombotic stroke subtype, reduced responsiveness to hypercapnia was found in 54 (49.1%) patients, in the cardioembolic subtype in 23 (20.9%), in the lacunar subtype in 8(7.3%) and in stroke of unspecified genesis in 5 (4.5%) patients. In the group of patients with early attacks there was also no correlation with a decrease in vascular reactivity in the carotid pools in patients with only acute symptomatic attacks in 18 (16.3%) and in patients in whom attacks recurred even one week after stroke onset in 18 (16.3%) patients ($\chi2=0.66$; $p=0.41$).

Focal pathological changes on EEG were registered more frequently in 44 (67.7%) patients with PTE and SSE with the development of epileptic seizures and decreased reactivity to hypercapnia, than in 27 (41.5%) patients with normal values of reactivity in carotid basins ($\chi2=8.8$; $p=0.003$). At the same time, pathological activity on EEG was registered predominantly in 49 (75,4%) patients with hypercapnia lower than 19%, while pathological activity was found in 29 (44,6%) patients with normal or slightly decreased (up to 20%) reactivity values ($\chi2=14,7$; $p=0,00013$). A comparative analysis of cerebrovascular reactivity in patients who suffered from CMP with the development of epileptic seizures showed that impaired perfusion reserve predominated in the vertebro-basilar basin in 94 (85.45%) patients compared to the carotid basin in 67 (60.9%) patients, $p<0.001$), with clearer differences observed among patients with grossly reduced perfusion reserves: a decrease in cerebrovascular reactivity of less than 10% in the vertebro-basilar basin was observed in 37 (33.6%) observations and in the carotid basin in 9 (8.2%, $p<0.001$). In the control group of patients there was no significant difference in changes of cerebrovascular reactivity in the carotid system and vertebro-basilar basin in 32 (66,7%) patients respectively. In assessing the state of hemodynamic indices, it is noteworthy that pulse indices in the left carotid pool were changed more frequently in patients with ischemic stroke with the development of epileptic seizures (in 23 (48%) observations increased, in 3 (6.2%) decreased, and in 21 (43.7%) normalized. Similarly, no differences in pulse index values in the right carotid pool were found for both sexes in the study and comparison groups ($\chi2=3,2$; $p=0,20$): increased pulse indexes were registered in 60 (54,5%) patients in the study and 22 (45,8%) in comparison groups, reduced - in 8 (7,3%) of the study and 2 (4,2%) comparison groups, normal values were registered in 42 (38,2%) of the study and 23 (47,9%) comparison group patients.

After functional tests we found that autoregulation on the right side was changed more frequently in patients with PTE and CVD in 29 (44,6%) patients of the main group than in comparison group patients in 8 (16,7%), $p<0,01$). The estimation of autoregulation state on the left side also revealed predominant changes of parameters in the patients of the main group in 46 (41,8%) patients compared to the comparison group patients in 12 (25%), $p<0,05$).

Among the patients studied 104 patients received 2 antiepileptic drugs, 45 patients received monotherapy. The course included the following neuroprotective drugs: Cytoflavin intravenously by drop 10 ml per day and Dinar by 4 ml intramuscularly for 10 days, then Dinar in tablet 250 mg 3 times

daily for 6 weeks, then Espa-Lipon in capsules 600 mg 3 times daily for 4 weeks. In parallel with neuroprotection, AED therapy was continued according to the regimen previously given to these patients. The efficacy of the corrective therapy was evaluated by the positive dynamics of clinical, hemodynamic and TCDG and EEG parameters in PTE patients in different periods after corrective treatment (after 1, 3, 6 months). The persistent effect of the corrective treatment course with the use of cavinton and Dinar in PTE patients within four months is shown.

A positive clinical dynamics was revealed - seizure frequency decreased by 50% or more in 92.5% of patients ($p<0.05$), including full control of seizures in 23.1 and 57.1% of PTE patients and a decrease by more than 75% in 44.9 and 33.3% of observations, respectively. The percentage of complete seizure control predominated in LVE patients ($p<0.05$). Six months after therapy, seizure frequency returned to baseline (Table 3)

Table 3.

Dynamics of epileptic seizures in PTE patients before and after corrective treatment

Indicators	Patients with post-traumatic epilepsy															
	Group I								Group II							
	Before the treatment		Through						Before the treatment		Through					
			1 month		3 month		6 month				1 month		3 month		6 month	
	n	%	n	%	N	%	n	%	n	%	N	%	N	%	n	%
Complete control of ES	-	-	25	22,7	23	20,9	-	-	14	29,2	27	56,2	25	52,1	16	33,3
decrease in the number of FTEs > 75%	32	29,1	49	44,5	45	40,9	35	31,8	23	47,9	16	33,3	17	35,4	18	40
decrease in the number of FTEs by 50%	32	29,1	23	20,9	25	22,7	37	33,6	11	22,9	5	10,5	6	12,5	14	29,2
decrease in the number of ES by 25%	39	35,5	13	11,8	17	14,5	34	30,9	-	-	-	-	-	-	-	-
no effect	8	7,3	-	-	-	-	4	3,7	-	-	-	-	-	-	-	-
Total	110	100	110	100	110	100	110	100	48	100	48	100	48	100	48	100

A reduction in subjective and objective neurological symptoms ($p<0.05$). The percentage of patients with regression of headache, dizziness, positive neurological dynamics was higher among PTE and SWD patients ($p<0.05$), and positive dynamics of reflex sphere and coordinator disorders was higher among PTE patients ($p<0.05$). This may be due to an improvement in cerebral haemodynamics.

Doppler-phase examination with functional tests performed one month after corrective therapy revealed positive dynamics of IVMR in all PTE patients ($p<0.05$) due to increased vasodilator reserve (hypercapnic Cr+ test) ($p<0.05$).

However in PTE group I patients, despite their positive dynamics, Kp+ values did not reach control values ($p<0.05$). No dynamics of hypercapnic test values were observed in GZ patients with BCA pathology indicating the deficit of vasodilator reserve ($p<0.05$). The increase of hypocapnic coefficient (Kr-), reflecting increased constrictor reserve, was seen in LVE patients and in patients with GZ pathology of BCA ($p<0.05$). The absence of its dynamics in MVE patients suggested a depletion of the

vasoconstrictor effect. The overshoot coefficient statistically significantly increased in PTE patients of all studied groups, which could indicate an increased vasodilation reserve after the course of corrective therapy. The data obtained 3 months after corrective therapy were similar; after 6 months, the resistive indices approached the initial data. Positive clinical, TCDG and haemodynamic effects of corrective treatment were most pronounced in LVE (Table 4), which was consistent with the baseline data, which were also higher compared to those in MVE patients and were explained by the prevalence of haemodynamically significant and insignificant vascular pathology in the BCA.

Table 4.

Dynamics of cerebrovascular reactivity in PTE patients at three and six months after corrective treatment

TDMC coefficients	PTE patients				Control group
	Patients with PTE + CVD		Patients with PTE		
	before treatment	after 1 month	before treatment	after 1 month	
IIWR	35,9±2,7	47,8±2,7	39,7±2,7	57,2±2,6	71,9±4,1
Cr+	1,15±0,02	1,18±0,02	1,15±0,02	1,28±0,02	1,36±0,06
Cr-	0,21±0,03	0,30±0,03	0,26±0,03	0,29±0,03	0,36±0,04
KO	1,18±0,03	1,22±0,03	1,12±0,02	1,25±0,02	1,26±0,04

The previously established correlation between IVMR indices and index of paroxysmal activity suggests that the increased compensatory capacity of the cerebral circulation reduces seizure readiness of the brain, affecting epileptogenesis and the frequency of epileptic seizures in temporal lobe epilepsy patients. Negative dynamics of clinical and neurophysiological parameters and approaching their baseline data 6 months after corrective treatment justify the need for repeated (at least twice a year) courses of corrective therapy in RE patients.

CONCLUSION: Thus, integrated therapy with AEDs and neuroprotective drugs significantly increases the efficacy of drug treatment of post-traumatic epilepsy in its pharmacoresistant course, which is objectively confirmed by the results of clinical and instrumental high-sensitivity research methods.

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