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EARLY CLINICAL-NEUROLOGICAL INDICATORS OF PALLIDAR PARKINSONISM

Аннотация:Данная научно-исследовательская работа посвящена выявлению и анализу клинико-неврологических показателей паллидарной недостаточности на ранних стадиях, его сравнительной диагностике, развитию ранних характерных симптомов заболевания на основе статистического анализа.

Ключевые слова: паркинсонизм, ранняя диагностика

Abstract: This research work deals with the identification and analysis of clinical neurological indicator of pallidar parkinsonism in the early stages, its comparative diagnosis, development of early characteristic symptoms of the disease based on statistical analysis.

Key words: parkinsonism, early diagnosis

Annotatsiya: Ushbu tadqiqot ish palidar etishovchilikni ertangi bosqichlarda klinik-nevrologik ko'rsatkichlarini aniqlash, taxlil qilish, uning qiyosiy tashxisi, statistik tahlillar asosida kasallikning ertangi xarakterli simptomlarini ishlab chiqish haqida so’z yuritiladi.

Kalit so’zlar: parkinsonizm, ertangi diagnostika

Introduction. Due to and thanks to improvements in quality of life, the average life expectancy has increased. Along with this, the number of patients suffering from cardiovascular diseases has also risen. Cerebral vascular atherosclerosis is the most common brain pathology. In many cases, this chronic process leads to the development of localized or diffuse encephalopathy with various clinical manifestations. The next most significant condition is atherosclerotic parkinsonism. The prevalence of these diseases is rapidly growing. (1,8).

Patients with atherosclerotic parkinsonism, depending on the pathological condition of the cerebral vessels, belong to the group of late-stage (chronic) encephalopathy or stage III dyscirculatory encephalopathy (DEP). Despite advancements in clinical neurological diagnostics, there are still certain challenges in detecting cerebrovascular diseases at early stages. The main reason is the absence of pronounced brain pathology in most patients at stages I–II of DEP.(2,3)

Objective of the Study. The aim of this study is the early detection, prediction, and prevention of pallidal insufficiency in patients with stage I–II DEP of atherosclerotic origin through a comparative assessment of clinical and neurological data.

Materials and Methods. The study included 45 patients: 22 men and 23 women aged 42 to 72 years (mean age of men — 62.2 ± 1.3 years, women — 64.5 ± 1.3 years). The diagnosis was established based on the classification of cerebrovascular diseases. The study was conducted at the clinic of Andijan State Medical Institute, where patients diagnosed with stage I, II, and III DEP of atherosclerotic origin underwent inpatient treatment.The first group included 21 patients with stage I–

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II DEP. The second group consisted of 24 patients with stage III DEP who exhibited symptoms of pallidal insufficiency.(1,5).

All patients underwent a standard clinical and neurological examination. In addition, an in-depth clinical assessment of the extrapyramidal system was conducted by analyzing motor and autonomic functions. The evaluation included gait changes, the presence of upper limb synkinesia, postural disturbances, handwriting changes, hypomimia, tremor, oligokinesia, plastic changes in muscle tone, as well as the condition of the muscles in the arms, legs, axial, and cervical regions.(3).

Results and Discussion. A comparative analysis of the clinical and neurological examinations of patients with stage I, II, and III DEP of atherosclerotic origin revealed that asthenic symptoms predominated over organic neurological signs. Among the primary clinical symptoms of DEP, general cerebral manifestations were identified, including headaches, dizziness, tinnitus, irritability, and others.

Additionally, patients exhibited pronounced sleep disturbances, such as shallow sleep with intermittent awakenings, prolonged wakefulness periods, and a feeling of fatigue upon waking. During the day, they experienced reduced alertness and frequent drowsiness.(7,8).

Patients also reported persistent fatigue, decreased work capacity, and heightened emotional excitability. Apathy, obsessive thoughts, and memory impairment—particularly affecting recent events—were commonly noted and often became the primary complaints. Clinical and neurological examinations of the second group indicated that patients with signs of vascular parkinsonism in stage III DEP experienced headaches, dizziness, tinnitus, fatigue, and memory impairments more frequently and with greater intensity compared to patients in the first group ($p < 0.001$). In contrast, asthenic disorders such as irritability, tearfulness, and sleep disturbances were significantly more pronounced in the first group ($p < 0.001$).(2,5).

Table 1. Primary Clinical Symptoms in Patients with Atherosclerotic Dyscirculatory Encephalopathy (DEP) Stages I, II, and III

№	Indicators	Group I 22		Group II 23		P
		Abs	%	abs	%	
	Headache	12	53,7±6,8	22	95,2±3,3	<0,001
	Dizziness	13	61,1±6,6	22	95,2±3,3	<0,001
	Noise in the head	12	53,7±6,8	19	83,0±5,8	<0,001
	Nervousness	16	72,2±6,1	23	100,0±0,0	<0,001
	Sleep disturbances	10	48,1±6,8	17	73,8±2,4	<0,01
	Negligently	12	53,7±6,8	23	100,0±0,0	<0,01
	Memory loss	14	63,0±6,6	20	92,9±4,0	<0,001
	Increased tearing (Lacrimation)	9	42,6±6,7	16	69,0±7,1	<0,01
	Fatigue	21	96,3±2,6	23	100,0±0,0	<0,001

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	Paresthesia	5	22,2±5,7	17	71,4±7,0	<0,01
	Photopsia	6	29,6±6,2	18	78,6±6,3	<0,01
	Tremor	-	-	15	64,3±7,4	<0,001
	Rigidity	11	50,0±6,8	22	97,6±2,4	<0,01
	Difficulty initiating movements	11	50,0±6,8	22	97,6±2,4	<0,01
	Changes in gait	2	11,1±4,3	20	85,7±5,4	<0,001
	Fading speech			13	54,8+7,7	<0,001

Note: P – statistically significant compared to data from Group I.

In-depth studies of patients with stages I–II of discirculatory encephalopathy (DCE) showed that even at the early stages of the disease, they reported the following complaints: rigidity (50.0 ± 6.8%), difficulties at the beginning of movement (50.0 ± 6.8%), gait changes (11.1 ± 4.3%), and stooping while walking (5.6 ± 3.1%).

Patients in the second group, suffering from vascular parkinsonism, mainly complained of: limited movement (88.1 ± 5.4%), difficulties at the beginning of movement (35.7 ± 7.4%), gait changes (85.7 ± 5.4%), muffled speech (54.8 ± 7.7%), and tremor of hands and legs (14.3 ± 7.4%). (4,8).

A comparative analysis of the objective neurological status of patients with stage I–II atherosclerotic DEP revealed the following disturbances: oculomotor nerve paresis (1.9 ± 1.8%), reduced convergence (48.1 ± 6.8%), and central paralysis of cranial nerves VII–XII. In 19 patients, anisoreflexia of tendon reflexes was observed, 5 on the right side and 13 on the left. All patients (100.0%) showed oral automatism reflexes, most often the Marinesco-Radovici reflex.(2)

Neurological examinations showed pathological reflexes in the arms and legs in 48.1 ± 6.8% of patients. Coordination tests (in the Romberg position) revealed inconsistencies in movements and unsteadiness. Detailed data are presented in Table 2.

In patients of the first group (stage I–II DCE), signs of extrapyramidal insufficiency were identified at early stages of the disease during preventive check-ups or by family observations, although the patients themselves did not attach importance to these symptoms. Gait changes (50.0 ± 6.8%) manifested as a slowdown in the pace of voluntary movements.

Gait disturbances were characterized by unsteadiness while walking, difficulties at the beginning of movement (50.0 ± 6.8%), sometimes slight pulsation (19.8 ± 1.8%), rigidity (51.9 ± 6.8%), and akinetic-hyperkinetic conditions. Changes in body position (50.0 ± 6.8%), hypomimia (29.6 ± 6.2%), decreased voice tone, and nasal speech (a change in voice timbre due to soft palate paresis in its initial part) (63.0 ± 6.6%) were also observed in patients.(5,8).

Table 2. Main indicators of objective neurological symptoms in patients with atherosclerotic genesis of dyscirculatory encephalopathy in stages

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I, II, and III.

№	Indicators	Group I (22)		Group II (23)		P
		Abs	%	abs	%	
1	Impaired convergence	11	48,1±6,8	20	88,1±5,0	<0,01
2	VII-XII central paralysis of cranial nerves	8	38,9±6,6	16	69,0±7,1	<0,01
3	Hypomimia	6	29,6±6,2	15	64,3±7,4	<0,001
4	bulbar palsy	14	63,0±6,6	21	90,5±4,5	<0,05
5	Pseudobulbar palsy	4	20,4±5,5	6	28,6±7,0	<0,01
6	Anisoreflexia	7	33,3±6,4	16	71,4±7,0	<0,01
7	Plastic tone	-	-	23	100,0±0,0	>0,05
8	Pulsation	1	2,9±1,8	8	35,7±7,4	<0,01
9	Bradykinesia	-		12	52,4±7,7	<0,001
10	Rigidity	11	51,9±6,8	23	100,0±0,0	<0,001
11	Akinesia	21	50,0±6,8	23	100,0±0,0	<0,001
12	Postural disturbances	21	50,0±6,8	23	100,0±0,0	<0,001
13	Tremor	-	-	15	64,3±7,4	<0,001
14	Micrographia	-	-	7	31,0±7,1	<0,01
15	Oral automatism reflexes	22	100,0±0,0	23	100,0±0,0	<0,001
16	Coordination disorders	5	24,1±5,8	21	85,7±5,4	<0,001
17	Memory decline	11	46,3±6,8	22	90,5±4,5	<0,01
18	Emotional lability	19	87,0±4,6	23	90,5±4,5	<0,001

P — statistically significant compared to data from Group I

The study did not reveal significant changes in sensory function. A comparative analysis of objective neurological examinations showed that, unlike the first group, patients in the second group exhibited early focal neurological symptoms, such as oculomotor nerve (III pair) impairment leading to gaze paralysis (21.4 ± 6.3%), reduced convergence (88.1 ± 5.0%), central paralysis of the facial nerve (VII pair) (69.0 ± 7.1%), bulbar paralysis (90.5 ± 4.5%), and hypomimia (64.3 ± 7.4%)

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Compared to Group I patients, those in Group II displayed pronounced organic neurological disorders, manifesting as pyramidal insufficiency ($p < 0.001$). Patients in the second group exhibited anisoreflexia of tendon reflexes (71.4 ± 7.0%) and oral automatism reflexes (100.0%), with Marinesco-Radovici, Chvostek, and Bechterew reflexes being the most common. Pathological reflexes in the arms and legs (81.0 ± 6.1%) included positive Rossolimo and Babinski reflexes in both upper and lower limbs. (4,5).

Patients demonstrated coordination disorders (85.7 ± 5.4%) in combination with pyramidal insufficiency ($p < 0.001$). Postural instability was observed in the Romberg test, and unsteady movement was noted in coordination tests

Patients in the second group also exhibited extrapyramidal dysfunction, characterized by muscle tremors and plastic hypertonia. Notably, 100% of patients showed increased plastic muscle hypertonia, which began in the distal parts of the body and progressed upwards. Muscle rigidity (100.0%) and bradykinesia (52.5 ± 7.7%) were observed together. The classic "cogwheel rigidity" symptom was present in all patients. Resting tremor was detected in only 14% of patients, primarily affecting the distal parts of the body. (6).

Patients in the second group showed distinct signs of gait disturbances, including slowness, tremor (100%), shortened steps, postural instability with frequent swaying, and difficulty turning the body. Their postural abnormalities were not only vertical but also horizontal, with a pronounced flexed posture (100%)

Analysis of the main clinical data and neurological status of patients with atherosclerotic genesis of stage II and III DEP leads to the following conclusions:

1. The primary risk factors leading to the development of vascular parkinsonism syndrome in the examined patients are cerebral atherosclerosis and arterial hypertension.

2. All signs of vascular parkinsonism, such as difficulty initiating movement, rigidity, body posture changes, slight hypomimia, and reduced speech modulation, already manifest in stage II of DEP with an atherosclerotic genesis.

3. In patients with stage III DEP, the characteristic features of vascular parkinsonism are: early development of postural instability in 100%, rigidity, hypomimia; 14% of patients exhibit increased muscle tone of the plastic type, tremor of distal parts of the body, and a flexor posture.

4. Characteristic features for patients with stage II and III DEP include combinations of symptoms of cerebral atherosclerosis (bulbar and pseudobulbar paralysis, pyramidal insufficiency, brain symptoms) and signs of vascular parkinsonism, which develop against the background of progressing cerebrovascular deficiency.(6,8).

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