

CLINICAL RESEARCH

## Cognitive Status in Patients with Chronic Cerebral Ischemia

Yokudhon N. Madjidova, PhD, ScD<sup>1\*</sup>; Durдона D. Usmanova<sup>2</sup>

<sup>1</sup>Tashkent Medical Pediatric Institute

<sup>2</sup> Tashkent Institute of Postgraduate Medical Education  
Tashkent, Uzbekistan

### Abstract

**The aim** of our study was to examine the cognitive functions in patients with chronic cerebral ischemia (stages I- II of discirculatory encephalopathy) of various origins. Systematization of the patients was performed according to EV Schmidt's classification of the vascular lesions of the brain. All the subjects were categorized into two groups. Group 1 consisted of 115 patients (42 men and 73 women) with chronic cerebral ischemia (CCI) that had developed, mainly, against the background of arterial hypertension (AH). Group 2 consisted of 122 patients (33 men and 89 women) with CCI, which developed, mainly, against the backdrop of atherosclerosis of the cerebral vessels. The mean age was  $54.2 \pm 0.7$  years in Group 1 and  $56.8 \pm 0.8$  years Group 2, respectively. Control group included 30 healthy subjects (mean age:  $52.2 \pm 0.9$  years) without any objective manifestations of CCI. The stage of cognitive deficit was determined by employing the MMSE test and the Bourdon test. The "Schulte Tables" technique was used for estimating the stability of attention and rate of sensorimotor reactions. Luria's Memory Ten-Word Retrieval Test (LMTWRT) was applied for estimating attention and memory.

The present study indicates that the cognitive deficits detected in patients with CIC were characterized by the greatest severity against the background of AH. AH predominantly damages the subcortical structures, resulting in subcortical angioencephalopathy, which ultimately leads to a deterioration of the intellectual-mental processes.

**Keywords:** chronic cerebral ischemia; arterial hypertension; of the cerebral vessels, cognitive deficits.

### Introduction

Cerebrovascular disease is one among the major problems encountered in modern medicine [1, 2]. It is well known that over the recent years the structure of vascular diseases of the brain has changed with the increase in the ischemic forms. This is a result of the steady increase in arterial hypertension and atherosclerosis, which are the major causes of cerebrovascular disease. In the study of certain forms of cerebrovascular accident, chronic ischemia was found to have the highest prevalence [3, 4, 5, 6].

Cognitive impairment is the earliest and most common manifestation of chronic cerebral ischemia (CCI). Thus, according to A.B. Lokshina and N. N. Yahno, cognitive

impairments are present in CCI stage I-II, in nearly 90% of the cases [2]. Cognitive impairment is a sufficiently reproducible manifestation of CCI. In repeated studies, it has manifested the same symptoms, even when the exam was conducted by different specialists. When compared with other neurological symptoms this is the essential difference of cognitive impairment in the early stages of CCI, the reproducibility of which is low [7-8]. Therefore, currently, there is a strong belief that the identification of cognitive impairments, which are characterized by several special features, has the greatest diagnostic value in patients with CCI [4, 6, 9].

**The aim** of our study was to examine the cognitive functions in patients with CCI (stages I- II of discirculatory encephalopathy) of various origins.

### Material and Methods

The study included 237 patients with CCI. Systematization of the patients was performed according to EV Schmidt's classification of the vascular lesions of the brain. All the subjects were categorized into two groups. Group 1

\*Corresponding author: Prof. Yokudhon N. Madjidova, MD, PhD. Head of Department of Neurology, Pediatric Neurology and Medical Genetics of Tashkent Medical Pediatric Institute. Tashkent, Uzbekistan. E-mail: [madjidova1@yandex.ru](mailto:madjidova1@yandex.ru)

consisted of 115 patients (42 men and 73 women) with CCI that had developed, mainly, against the background of arterial hypertension (AH). Group 2 consisted of 122 patients (33 men and 89 women) with CCI, which developed, mainly, against the backdrop of atherosclerosis. In both groups, a predominance of women was noted. The mean age was  $54.2 \pm 0.7$  years in Group 1 and  $56.8 \pm 0.8$  years Group 2, respectively. Control group included 30 healthy subjects (mean age:  $52.2 \pm 0.9$  years) without any objective manifestations of CCI.

The exclusion criteria were age less than 35 and older than 75 years, CCI stage III, other etiologies of encephalopathy, stroke, diabetes, epilepsy, organic diseases of the brain and spinal cord (the hereditary, demyelinating, and degenerative diseases, tumors), blood diseases and autoimmune diseases.

The stage of cognitive deficit was determined by employing the MMSE (Mini-Mental State Exam) test and the Bourdon test. The "Schulte Tables" technique was used for estimating the stability of attention and rate of sensorimotor reactions. Luria's Memory Ten-Word Retrieval Test (LMTWRT) was applied for estimating attention and memory.

Statistical analysis was performed using the statistical software «Statistica». For data with normal distribution, intergroup comparisons were performed using student's t-test. The mean (M) and standard error (SE) of the mean were calculated. The difference was considered reliable when  $P < 0.05$ .

## Results and Discussion

The data presented in Table 1 showed that the MMSE score was  $29.5 \pm 0.1$  points in the control group. In Group 1 patients this score was  $19.7 \pm 0.2$  ( $P < 0.001$ ) which corresponded to advanced cognitive impairment, in Group 2 it was  $21.8 \pm 0.2$  ( $P < 0.001$ ), which corresponded to mild cognitive impairment. Differences in the MMSE score between the groups were statistically significant.

**Table 1**

### Mini-Mental State Examination

Parameter	Control group (n=30)	Group 1 (n=115)	Group 2 (n=122)
MMSE score (Norm - 30)	$29.5 \pm 0.1$	$19.7 \pm 0.2^{***}$	$21.8 \pm 0.2^{***\wedge\wedge}$

\*\*\* -  $P < 0.001$  vs control;

$\wedge\wedge$  -  $P < 0.001$  between Group 1 and Group 2

The analysis of cognitive function according to the "Schulte's Tables" technique is presented in Table 2. In the control group, the mean time for task performance was  $31.0 \pm 0.4$  sec. Time indices were  $49.4 \pm 0.3$  sec and  $41.2 \pm 0.3$  sec, in Groups 1 and 2, respectively ( $P < 0.001$ ).

**Table 2**

### Cognitive function according to the "Schulte's Tables" technique

Parameter	Control group (n=30)	Group 1 (n=115)	Group 2 (n=122)
"Schulte's Tables", sec	$31.0 \pm 0.4$	$49.4 \pm 0.3^{***}$	$41.2 \pm 0.3^{***\wedge\wedge}$

Abbreviations as in Table 1.

Therefore, it was observed that the rate of the sensorimotor reactions and attention differed from the control. Patients in both groups demonstrated a slowing down in the pace of the sensorimotor reactions and a decrease in the speed of switching attention, more pronounced in the Group 1 patients. This confirms the fact that the AH insidiously damages the subcortical structures and impairs the intellectual and mnemonic processes, which in turn leads to a slowdown in the pace of the sensorimotor reactions and reduces the speed of switching attention.

The results of the Luria's Memory Ten-Word Retrieval Test (LMTWRT) are presented in Table 3. In the control group, the short-term memory was  $7.7 \pm 0.1$  words, long-term memory -  $8.9 \pm 0.1$ , and the productivity of memorization was  $87.3 \pm 0.3$ . The short-term memory indices were  $5.0 \pm 0.1$  words in Group 1 patients and  $5.5 \pm 0.1$  in Group 2 patients, respectively. The long-term memory indices were  $5.5 \pm 0.1$  words in Group 1 patients and  $6.7 \pm 0.1$  in Group 2 patients. The productivity of memorization was  $63.1 \pm 0.3$  in Group 1 and  $67.4 \pm 0.3$  in Group 2. Thus, the LMTWRT revealed a decline in both the short and long-term memory parameters and the productivity of memorization in both groups. Thus, in Group 1 patients, we found a decline in the short-term memory by 35.1%, long-term memory by 38.2%, and the productivity of memorization by 27.7% compared with the control group. These parameters were also seen to be decreased in Group 2 patients: short-term memory by 28.5%, long-term memory by 24.7% and the productivity of memorization by 22.8% that was much less when compared with Group 1 patients.

**Table 3**

### The results of the Luria's Memory Ten-Word Retrieval Test

Group	Short-term memory	Long-term memory	Productivity of memorization
Control group (n=30)	$7.7 \pm 0.1$	$8.9 \pm 0.1$	$87.3 \pm 0.3$
Group 1 (n=115)	$5.0 \pm 0.1$ 35.1%	$5.5 \pm 0.1$ 38.2%	$63.1 \pm 0.3$ 27.7%
Group 2 (n=122)	$5.5 \pm 0.1$ 28.5%	$6.7 \pm 0.1$ 24.7%	$67.4 \pm 0.3$ 22.8%

% - percentage change in relation to control.

The Bourdon test revealed a decline in the concentration and stability of attention in both the groups (Table 4). In the control group, the concentration and stability of attention were  $516.0 \pm 20.1$  and  $4.4 \pm 0.05$  points, respectively. In Group 1 patients, these parameters were  $124.6 \pm 4.1$  and  $3.4 \pm 0.03$  vs  $170.8 \pm 5.6$  and  $3.7 \pm 0.02$  in Group 2 patients ( $P < 0.001$ ).

**Table 4.**

### Concentration and stability of attention in the Bourdon test

Parameter	Control group (n=30)	Group 1 (n=115)	Group 2 (n=122)
Concentration of attention	$516.0 \pm 20.1$	$124.6 \pm 4.1^{***}$	$170.8 \pm 5.6^{***\wedge\wedge}$
Stability of attention	$4.4 \pm 0.05$	$3.4 \pm 0.03^{***}$	$3.7 \pm 0.02^{***\wedge\wedge}$

Abbreviations as in Table 1.

The significant decline in the concentration and stability of attention in Group 1 patients can be explained as a consequence of the early and progressive destruction of the white matter of the brain, in hypertension.

Thus, it can be assumed that the cognitive impairment in patients with CCI are caused by the phenomenon of cortical-subcortical and cortical-cortical dissociation (disconnection syndrome), which were often detected in those with CCI [2, 4]. In the disconnection syndrome, lesions of the association pathways between the various parts of the brain are observed, that lead to the delay and reduction of the intellectual-mental functions. The leading role in this pathology, in most cases, belongs to the lesion of the white matter of the brain, especially related to the association pathways between the frontal lobes with the other structures of the central nervous system.

## Conclusion

The present study indicates that the cognitive deficits detected in patients with CIC were characterized by the greatest severity against the background of AH. AH predominantly damages the subcortical structures, resulting in subcortical angioencephalopathy, which ultimately leads to a deterioration of the intellectual-mental processes.

## References

1. Zakharov VV, Yahno NN. Syndrome of mild cognitive impairment in the elderly and senile age. *Russ Med Zh* 2004; 10:573-576. [Article in Russian].
2. Yahno NN, Zakharov VV, Lokshina AB. Syndrome of mild cognitive impairment in vascular encephalopathy. *Zh Nevrol Psikiatr Im SS Korsakova* 2005; 2:13-17. [Article in Russian].
3. Zakharov VV. Atherosclerosis of cerebral and peripheral arteries: treatment issues. *Russ Med Zh* 2007; 10:833-836. [Article in Russian].
4. Parfenov VA. Hypertension and stroke. *Nevrol Zh* 2001; 6(6):4-7. [Article in Russian].
5. Preobrazhenskay IS, Yahno NN. Vascular cognitive disorders - clinical manifestations, diagnosis and treatment. *Nevrol Zh* 2007; 12(5):45-50. [Article in Russian].
6. Petersen RC, Touchon J. Consensus on mild cognitive impairment. Research and practice in Alzheimer's disease. *ADS-ADCS joint meeting* 2005; 10:24-32.
7. Birks J, Grimley Evans J. Ginkgo biloba for cognitive impairment and dementia. *Cochrane Database Syst Rev* 2007; 18(2):CD003120.
8. Ihl R. The impact of drugs against dementia on cognition in aging and mild cognitive impairment. *Pharmacopsychiatry* 2003; 36 Suppl 1:S38-43.
9. Brandt J. Mild cognitive impairment in the elderly. *Am Fam Physician* 2001; 63(4):620-6.