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Issues of multipurpose forecasting of ischemic strokes development

Abdullaeva M.B[1] , Raimova M.M[2], Majidova Y.N[3], Azimova N.M[4]

[1]Senior Lecturer of the Department of Neurology and Physiotherapy, Tashkent State Dental Institute
[2]DSc, docent of the Department of Neurology and Physiotherapy, Tashkent State Dental Institute
[3]DSc, prof., Head of the Department of Neurology, Pediatric Neurology with Medical Genetics course, Tashkent Pediatric Medical Institute
[4]PhD, docent of the Department of Neurology, Pediatric Neurology with Medical Genetics course, Tashkent Pediatric Medical Institute

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Abstract

TIA is a recognized risk factor for stroke and, in turn, is associated with the same risk factors as stroke. The mechanism of TIA development is a focal decrease in blood supply to the brain tissue due to damage to the cerebral vessels. The pathogenetic difference between TIA and ischemic stroke is the reversibility and instability of the pathological process and organic disorders in TIA. Our studies have shown a polymorphism of the symptoms of the clinical picture of TIA. Analysis of clinical, medical history and clinical features of TIA revealed their dependence on a number of factors. In particular, these are etiological factors, the dependence of focal symptoms on the vascular pool in which transient ischemia occurred, the level of systolic blood pressure, which determined not only the polymorphism of symptoms, but also the duration of TIA itself.As our MRI studies showed, more often than not, ischemic stroke developed in patients with TIA in the strategic zone of the brain and multi-infarction conditions.

Keywords: ischemic stroke, TIA development, Cerebral vessels

Actuality

Transient ischemic attack (TIA), today, is the main symptom that signals the threat of a stroke. In 30-40% of patients after TIA, a stroke develops in the next 5 years. More than 20% of these strokes occur during the first month, and almost half - during the first year after TIA [3,5]. The risk of stroke is approximately 10% in the first year, and then about 5% annually. The likelihood of a stroke is higher with repeated TIA and an increase in the patient's age (the likelihood of a stroke increases by almost 1.5 times with an increase in age by 10 years) [6]. This requires therapeutic measures to prevent the development of AI in this category of patients. According to the literature, numerous cohort studies in the USA and other countries of the world show different incidence and prevalence of TIA, which ranged from 0.37 to 1.1 per 1000 people per year, among Americans the incidence is 2.3% [1,2]. With age, the incidence of TIA increases - from 3.4 to 4.6%. In a British study, the incidence of TIA increases with age, among people over 85 years of age reaches 6.41 per 1000 population. It should be noted that the history of TIA in patients with stroke is from 7 to 40%. In Russia, the diagnosis of TIA is made annually by 40 thousand people [4,5]. According to B.G. Gafurov in Uzbekistan, the incidence of AI is up to 40 thousand cases annually. These numbers can be much higher, since TIA is characterized by quick-passing clinical symptoms, which reduces the patient's appeal for medical care. TIA attacks can often recur or occur only once or twice. In many cases, patients do not attach transient short-term disorders of significant importance and do not seek the advice of a doctor, so it is difficult to assess the prevalence of TIA. Today, TIA is considered the same urgent condition as a stroke. Experts from the European Organization for the Control of Stroke (ESO) reviewed the management of such patients. TIA is a recognized risk factor for stroke and, in turn, is associated with the same risk factors as stroke. The mechanism of TIA development is a focal decrease in blood supply to the brain tissue due to damage to the cerebral vessels. The pathogenetic difference between TIA and ischemic stroke is the reversibility and instability of the pathological process and organic disorders in TIA. The leading role in the formation of TIA is played by atherosclerotic lesions of large and medium caliber arteries [1, 2, 5]. Mostly the

carotid and vertebral arteries are affected. The occurrence of TIA is based on the processes of destruction of atherosclerotic plaques, stenosis (atherosclerosis), obliteration of the lumen of the artery with a plaque, embolism, thrombosis, which complicates atherosclerosis in the plaque (atherothrombosis). Along with this, TIAs may be due to secondary structural changes in blood vessels associated with hemodynamic disturbances and vascular recalibration [1,5,6]

It is known that cognitive impairment of varying severity is detected in 40-70% of patients who have suffered a stroke. The prevalence of dementia in the first 3 months. after a stroke, it ranges from 5 to 32%, and after 12 months. - from 8 to 26% [154]. In the first 6 months. the risk of developing dementia is high and persists for several years. In clinical trials, the proportion of patients 1 year after a stroke is 9-17%, after 3 years - 24-28%, and by 5 years it increases to 32%. Those. 5 years after a stroke, the risk of developing dementia is 5 times higher than in the population [154, 208]. However, the presence of cognitive impairment, reaching in some cases up to the level of vascular dementia (VD) in patients with stage II chronic cerebral ischemia, which is up to 70-80%, according to some authors, is important. Considering that the morphological substrate for the development of diabetes is multiple heart attacks, microinfarctions, diapedetic hemorrhages, which can also be attributed to the development factors of acute forms of cerebrovascular diseases the common mechanisms of the development of ischemic stroke and vascular dementia. This, in turn, determines the importance of studying cognitive impairment in patients with pre-stroke forms of cerebrovascular diseasesand, in particular, with TIA.

Aim of the study:to study the prognostic value of cognitive impairment in the development of ischemic stroke in patients with TIA on the backgroundof chronic cerebral ischemia (CCI).

MATERIALS AND METHODS

To solve the set scientific goals and objectives, we studied 64 patients with various forms of cerebrovascular pathology (CVP) who were treated in the neurological department of the 3-TMA clinic. Of these, group I (main) - 30 patients with TIA on the background of chronic cerebral ischemiaII stage and group II (comparisons) - 34 patients with ischemic stroke, after TIA. It should be noted that patients of both groups were in the early or late recovery, or residual periods of stroke. The control group consisted of 20 persons of identical age who did not have signs of acute cerebrovascular pathology or cognitive impairment. The follow-up period was 5 years.

When distributed by sex, we revealed a predominance of males in a ratio of 1.5: 1. The average age of patients was 68.3 ± 13.2 years.

Research methods included clinical and neurological research, biochemical methods: a coagulogram of blood, blood for cholesterol and its fractions, MRI of the brain, ultrasound examination of cerebral vessels.

RESULTS

During the studying of anamnesis of patients with TIA and ischemic stroke after the TIA, we revealed some clinical features of these patients. In our observations (both groups of patients), TIAs were most often observed with a frequency of 1 time in 6 months and 1 time per year (52.6%). TIAs were observed less frequently with a frequency of once a month (25%) and somewhat less frequently with a frequency of 1 time per week (21.25%). It is important to note that patients of these groups were under closer supervision by doctors, due to the greater risk of developing of ischemic stroke. Upon further observation, most patients (55%) of the latter group developed an ischemic stroke within 48 hours.

The most common cause of the development of TIA and ischemic stroke in our observations was a combination of cerebral atherosclerosis and stageII hypertension. Moreover, more often, 45% of cases, we have seen cases of the development of TIA against the background of a combination of two etiological factors - cerebral atherosclerosis and stage II hypertension. This was the reason for studying the dependence of the development of TIA against the background of a combination of arterial hypertension and cerebral atherosclerosis. A study of the history of patients with TIA, the latter, as a rule, developed against the background of hypertension. In this case, the average blood pressure at the time of TIA was 156.4 ± 12.7 mmHg and often regarded by primary care physicians as hypertensive cerebral crises. The prevalence in the clinical picture of focal symptoms over cerebral symptoms served as a differential diagnostic criterion characteristic of TIA. It is important to note that in the course of our studies, we revealed a dependence of the duration of the transient attack on the numbers of blood pressure. High systolic blood pressure had an effect on the duration of transient dyshemia. The presence of hypertension led to an increase in the duration of TIA, i.e. against the background of systolic blood pressure more than 160 mmHg and more, TIA lasted from 1 hour to a day. It should be noted that the number of patients with TIA on the background of high systolic blood pressure (more than 140-160) was one and a half times the number of TIA observations on the background of normal blood pressure (110-130). Another important factor in the development of TIA, in our opinion, is the experience of the disease in hypertension. In our observations, most patients suffered from hypertension for more than 10-15

years. The role of hypertension in the development of TIA can be explained by several pathogenetic mechanisms. Firstly, it is thromboembolism of small cerebral vessels, secondly, this persistent cerebral vasoconstriction due to hypertension, exacerbating chronic cerebral ischemia, thirdly, prolonged arterial hypertension (in most cases poorly corrected by medications) leads to disruption of the mechanisms of autoregulation of the vessels of the brain brain, which in turn reduces the compensatory mechanisms of cerebral blood flow and increases the risk of subsequent ischemic stroke.

Our studies have shown a polymorphism of the symptoms of the clinical picture of TIA. Analysis of clinical, medical history and clinical features of TIA revealed their dependence on a number of factors. In particular, these are etiological factors, the dependence of focal symptoms on the vascular pool in which transient ischemia occurred, the level of systolic blood pressure, which determined not only the polymorphism of symptoms, but also the duration of TIA itself. In addition, we determined the average duration of TIA in the carotid pool, which was on average more than 1 hour and up to a day with a frequency of up to 1 time in 6 months. In the clinical picture of TIA, depending on the vascular pool, either monocular blindness syndrome, characteristic of TIA in KB, or dizziness syndrome, characteristic of TIA in vertebrobasilar insufficiency (VBI), predominated. If the etiological factors of TIA in CB were more often the combination of asthenic syndrome and hypertension II, then in patients with TIA in VBI, in addition to these etiological factors, we noted pathological tortuosity of the vessels. In order to predict the significance of TIA in the development of ischemic stroke 34 patients with a history of AI having TIA were examined. When studying the anamnestic features of ischemic strokeafter TIA, we paid attention to the frequency and severity of the clinical picture of TIA. These aspects of TIA, as shown by our studies, determine the likelihood and severity of the course of ischemic stroke. All patients who had a history of stroke had TIA in the carotid or vertebro-basilar pool. Moreover, the prescription of TIA development ranged from an average of 1 year to 5 years. In 55.88% of the cases of our observations, ischemic strokes developed in the left carotid basin, which suggests their thrombotic origin and the prevalence of atherogenesis as the dominant etiological factor.

According to the anamnesis, up to 88.2% of patients after TIA took antihypertensive and antiplatelet agents affecting the rheological properties of blood for several months or a year. However, by the end of the first year, the number of patients receiving drug etiopathogenetic therapy is reduced to 20%, which can be considered the cause of their stroke.

The average period between TIA and subsequent stroke in our observations was 2.4 ± 1.3 years. The latter is of great importance in the prevention of stroke, i.e. It is during this time period that patients who have undergone TIA should be under the greater care of neurologists, which will reduce the risk of developing ischemic stroke.

Next, we studied the clinical and neurological features of TIA. Clinical and neurological features in the examined patientsare presented in Table 1.

Table 1: Clinical and neurological features in patients of 1st and 2ndgroups according to the results of our own observations

Group of patients	1st group (n=80)	2nd group (n=34)	3rd group (n=20)	P
Headachesanddizziness	87,5%	76,4%	60%	
Monocularblindness	46,25%	-	-	
Mono- or hemiparesis	17,5%	100		
Brachycephalic paresis	5%	-		
Cranialnervespalsies	-	100	40%	
Speechdisorders	21,25%	41,14		
Transient amnesia	8,75%	-		
Meningeal signs	-	17,6%		
Cognitive impairment	-	79,4%		

Most often (46.25%) in patients with TIA in the carotid pool, we met monocular blindness, which was accompanied by a decrease in vision or its complete loss on the side of the stenosed artery. 25% of patients complained of a feeling of "curtains" or "flaps" in the eye, which were sometimes provoked by bright sunlight or glare. In 10 patients (17.5%), we diagnosed optic-pyramidal syndrome, when short-term hemi- or monoparesis or hemihypesthesia on the contralateral side was observed against the background of monocular blindness.

In 3 cases (5%) there was a transient brachycephalic paresis with monoparesis of the arm and a slight central paresis of the facial nerve on the side opposite to the site of ischemia. In 12 patients (21.25%), TIAs were accompanied by episodes of mild aphatic disorders, which are described in the literature as cortical dysphasia.

Often, in 8.75% of cases, patients described transient amnesia, which, as a rule, was expressed in disorientation in space and time. In all cases, transient amnesia developed as TIA against the background of malignant arterial hypertension and passed several hours after the decrease in blood pressure. Transient mono- or hemiparesis was most often combined with monocular blindness or paresis of the facial nerve and were symptoms of optic pyramidal or brachycephalic paresis.

Next, we conducted an analysis of focal clinical symptoms in patients with ischemic stroke after TIA. All cases of ischemic stroke noted in our observations occurred in the carotid basin. Of the 34 cases of ischemic stroke, 21 occurred in the left carotid basin, and 13 cases in the right. Only in 4 cases did the ischemic strokepool not coincide with the TIA pool. This indicates the importance of TIA, not only in the aspect of forecasting the ischemic stroke itself, but also in determining the localization of the focus of ischemia. The variety of focal neurological symptoms depends on the last factor; the most common focal symptoms such as hemiparesis, hemigipesthesia, and speech impairment (88.23% of cases). In the structure of speech impairment, we noted motor aphasia (23.5%), sensory aphasia (17.64%), but the most common cases were mixed or total aphasia (47.05%). Muscle strength in paretic limbs averaged 2.6 ± 0.7 points and corresponded to paresis. Muscle tone on the paresis side, as a rule, was increased (61.76%) and only in 26.4% of cases we noted muscle hypotension in the acute period of AI. Pathology of the cranial nerves in the form of a central paresis of the facial and sublingual nerves was observed in 79.4%, and in 76.5% of the ischemic stroke proceeded with meningeal signs. In 82.35%, pyramidal insufficiency was expressed not only in the form of hemiparesis, but also with pathological stop signs.

Our studies have shown that most often (58.82%) of ischemic stroke develop 2-3 years after TIA. The smallest percentage (11.75%) of the incidence occurs 1 year after TIA, because during this period after a stroke are under more close supervision of doctors. Often (61.76%) the ischemic stroke pool is left carotid and in 88.32% of cases coincides with the pool in which the TIA occurred.

Of the subjective complaints, the most frequent (88.32%) were complaints of weakness and numbness in the limbs, which corresponded to the side of hemiparesis and hemihypesthesia. Other complaints include complaints of headaches and memory loss. In the structure of focal neurological symptoms, in our observations, hemisyndrome and speech disorders were often found (88.32% of cases). Of speech disorders, we noted motor aphasia (23.5%), sensory aphasia (17.64%), but the most common cases were mixed or total aphasia (47.05%).

Correspondence of the vascular basins of TIA and ischemic stroke after the transferred TIA indicates the atherothrombotic or embolic mechanism of persistent cerebral dyshemia and indicates the need for measures of primary prevention of stroke aimed at changing the rheological parameters of the blood, which also undergo certain changes.

Next, we studied the prognostic significance of cognitive impairment in the development of ischemic strokes after TIA. Analysis of anamnestic data carried out over the 5th period showed that in the II group of patients with the initial 23 cases of post-stroke cognitive impairment, by the 5th year the indicator increased to 29 cases, i.e. by 17.6%. Those. post-stroke vascular dementia was noted, stroke aggravated cognitive deficit. In group I patients, we noted in 47 cases the presence of cognitive impairment, which in 42 (52.5%) cases was regarded by us as manifestations of vascular dementia against a background of chronic cerebral ischemia. Of the indicated number of patients, all received therapy with the inclusion of nootropic and vascular drugs. But despite this, during the entire observation period (5 years), repeated TIAs developed in 17 (21.25%) cases, and ischemic stroke in 13 cases (16.2%). A comparison of baseline indicators of neurological status and cognitive sphere revealed baseline differences in these patients. So, in patients with a stroke that developed on the background of diabetes mellitus against the background of stage II chronic cerebral ischemia. Diabetes indicators were rougher, and the treatment did not include prolonged use of vascular drugs, and correction of cognitive impairment. Whereas in patients with TIA who developed on the background of diabetes against the background of stage II chronic cerebral ischemia. Patients received long-term and adequate antiplatelet therapy, with the addition of central cholinesterase drugs. Thus, we can conclude that the presence of uncorrected diabetes negatively affects the prognosis of ischemic stroke after TIA. Adequate complex therapy of cognitive impairment on the background of stage II chronic cerebral ischemia with TIA can be one way to prevent stroke after TIA.

During the entire observation period, positive dynamics was noted in both groups, while patients who underwent ischemic stroke showed a better recovery of cognitive functions on the MMSE scale than patients with TIA by 19.7% and 14.8%, respectively.

According to our studies, the cognitive functions assessed using the MMSE scale significantly differed between patients who underwent TIA and ischemic stroke. So, in patients with TIA, the average score on a scale was 24.6 ± 2.02 at the first visit, while in patients with ischemic stroke, this indicator was significantly lower and amounted to 20.94 ± 2.8. Among patients with IS, mild dementia prevailed - 50% (n = 17), and in the group with TIA, most patients had pre-dementive cognitive impairment - 70.5% (n = 24) (Table 2).

Table 2: Cognitive functions according to the MMSE scale

Indicators	**1st visit**	**After 9-10 days**	**After 3 months**
1st group (n=80) TIA	24,6±2,02	27,6±1,5	28,9±1,2
2nd group (n=34) ischemic stroke after TIA	20,94±2,8	24,1±2,16	26,1±1,9
Indicators	ПД	ЛД	УД
1st group (n=80) TIA	71,25% (n=67)	21,25% (n=17)	6,25% (n=5)
2nd group (n=34) ischemic stroke after TIA	14,7% (n=5)	50,0% (n=17)	35,3% (n=12)

As can be clearly seen from the presented Table 2, the initial values of cognitive function in patients of both groups were different. If the manifestations of cognitive deficit at the first examination in the first group was 24.6 ± 2.02, then by the end of 3 months he distilled the knowledge of the norm 28.9 ± 1.2. The dynamics of cognitive deficit indicators on the MMSE scale was worse. For 3 months it reached normal values of 26.1 ± 1.9 and corresponded to moderate cognitive impairment. Those. transient cerebral ischemia was accompanied by aggravation of cognitive impairment, which was reversible by 3 months after TIA.

Based on the foregoing, we can say the severity of cognitive impairment determined the duration and risk of stroke. That is, the more severe the cognitive impairment, the shorter the time interval between TIA and the subsequent stroke of ischemic stroke. Drug correction of existing cognitive impairment has led to an increase in the period between TIA and ischemic stroke. The latter, in our opinion, allows us to consider cognitive impairment as a risk factor for the development of ischemic stroke, and the degree of cognitive impairment as a predictor of brain damage. As noted above, the morphological substrate for the development of diabetes, according to the literature [154, 208], is multiple heart attacks, micro-infarctions, diapedetic hemorrhages, which can also be attributed to the development factors of acute forms of CVD, the common mechanisms of the development of ischemic stroke and vascular dementia on the background stage II chronic cerebral ischemia. In this regard, in order to clarify the morphological picture of the brain, patients of group I performed an MRI of the brain. In patients with cognitive impairment reaching the level of dementia, we revealed small focal heart attacks in the strategic zone (Figure 1) and multi-infarction states (Figure 2). In patients with moderate cognitive impairment, as a rule, an expansion of the ventricular system of the brain and expansion of the furrows were detected, which is regarded by us as a manifestation of chronic cerebral ischemia. It should be noted that frequent and near-term ischemic stroke TIA field developed in patients with TIA in the strategic zone and multi-infarction conditions.

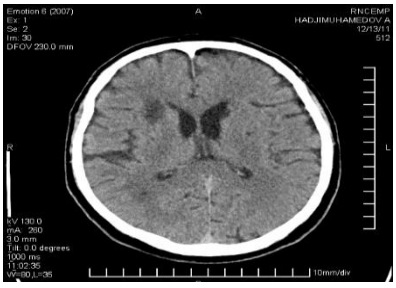

Figure 1: TIA in the strategic area of the brain (frontal lobe)

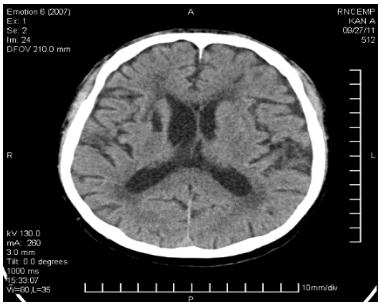

Figure 2: Multi-infarction condition

Summarizing the results of our studies, it can be noted that in addition to the known factors for the development of ischemic stroke after TIA, 2 more factors can be distinguished. In our opinion, this is the presence of a cognitive deficit, against which ischemic stroke develops, and its severity. In addition, the morphological state of the brain. As our MRI studies showed, more often than not, ischemic stroke developed in patients with TIA in the strategic zone of the brain and multi-infarction conditions.

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