Features of Neuroimaging Diagnostics of Transient Ischemic Attacks

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Abstract

Transient ischemic attacks (TIA), as precursors of cerebral strokes, occupy an important place among all forms of cerebrovascular insufficiency. Regarding epidemiology of transient ischemic attacks (TIA), there are no accurate data in most countries. In the United States, up to 5 million adult citizens per year suffer from these episodes, many TIA remain undiagnosed. These episodes of a sudden and short-term neurological deficit have long been considered as benign and harmless. Most general practitioners and patients misunderstand or insufficiently understand TIA's nature and significance, perhaps, this is the reason of insufficient medical interest, lack of statistical data on this nosological unit. Transient ischemic attacks (TIA) are clinically defined as rapidly developing focal and sometimes diffuse (cerebral) dysfunctions of the brain that are caused by local ischemia and last no longer than a day[1]. Over the past two decades, many views on TIA have changed significantly, approaches to the diagnosis and treatment of patients have become much more intense and aggressive. Modern knowledge about TIA is of paramount importance both for proper organization of patient care and educational programs among the population; their importance can hardly be overestimated.

Keywords: *Transient ischemic attack, Cardiomagnyl, ischemic stroke, prevention.*

Introduction

Transient ischemic attacks (TIA), as precursors of cerebral strokes, occupy an important place among all forms of cerebrovascular insufficiency. Regarding epidemiology of transient ischemic attacks (TIA), there are no accurate data in most countries. In the United States, up to 5 million adult citizens per year suffer from these episodes, many TIA remain undiagnosed. These episodes of a sudden and short-term neurological deficit have long been considered as benign and harmless. Most general practitioners and patients misunderstand or insufficiently understand TIA's nature and significance, perhaps, this is the reason of insufficient medical interest, lack of statistical data on this nosological unit. Transient ischemic attacks (TIA) are clinically defined as rapidly developing focal and sometimes diffuse (cerebral) dysfunctions of the brain that are caused by local ischemia and last no longer than a day [1]. Over the past two decades, many views on TIA have changed significantly, approaches to the diagnosis and treatment of patients have become much more intense and aggressive. Modern knowledge about TIA is of paramount importance both for proper organization of patient care and educational programs among the population; their importance can hardly be overestimated. TIA shall be considered an urgent situation for the following reasons: • first - the risk of ischemic stroke development after TIA is rather high; • second - effective secondary prevention is available. In this respect, early and reliable diagnosis of TIA is utterly important, allowing correct treatment of this pathology. In TIA diagnostics, ultrasonic Doppler examination and computer tomography are used in most

cases. Usually, TIA is an acute episode in the general clinical picture of chronic progressive cerebral ischemia, neuroimaging method also make it possible to detect dyscirculatory encephalopathy signs. Atherosclerosis is defined as the most common cause of TIA in Western Europe and America in 80-90% of cases. Atherosclerotic multiple lesions are more common than isolated ones. According to Spiridonov A.A. (1996), pathology of only one carotid artery occurs in 51.7% of cases. The lesion of one vertebral artery was observed in 38% of cases. The incidence of multiple BCA lesions ranges from 50 to 93.8%. BCA lesion in 33% of cases is located intracranially, or in the zone, inaccessible for direct surgical revision. Other 67% were extracranial, 52% of these were located in CCA bifurcation area, 20% in the area of vertebral artery mouths and 9% in the area of the main branches of the aortic arch.Pathological deformations of carotid and vertebral arteries takes the second place in the structure of TIA causes. The reasons for development of pathological deformations have not yet been determined. Most authors believe that they result from congenital (embryogenesis disorder) or acquired factors (weakening of the artery wall elastic structure, atherosclerotic artery lesions, age-related anatomical changes in the cervical spine), or functional adaptive mechanisms aimed at pulse wave reduction (with arterial hypertension) and blood flow uniformity.

The aortic arch syndrome (AAS) is another cause of BCA lesion. It is observed in approximately 10-15% of patients and is a polyetiological autoimmune vasculitis with a primary lesion of the aorta and main arteries of the elastic type. AAS affects people aged from 10 to 40 years, more often women[7,9,15]. AAS is characterized by multiple lesions of the aortic arch branches, often symmetrical, for example, two carotid and two subclavian arteries. Most often, the subclavian artery is occluded and almost twice as often - the left subclavian artery. Most often, a subclavian artery is occluded and almost twice as often - a left subclavian artery.

In 5-10% of cases, TIA is caused by relatively rare diseases, causing lesion (blockage or narrowing) of the BCA and resulting local brain ischemia. Acute and blunt vascular injuries with arterial thrombosis, BCA dissection, BCA fibromuscular dysplasia, as well as extravasal vascular compression.

Dissection of pre-cerebral and, less commonly, cerebral arteries is the cause of about 2% of TIA cases, it is more typical for young women. BCA and VA dissection can occur due to trauma, unsuccessful manipulation of the cervical spine or developmental abnormalities. A hematoma, formed under the intimal artery, causes its lumen narrowing and can cause thrombosis or embolism.

The clinical picture of TIA is very diverse, but with this pathology there are no clear pathognomonic symptoms, their presence would help clinicians in diagnositics nd timely selection of treatment tactics for detected lesions, including surgical revision of pathology. If TIA is located in the carotid pool, then, as a rule, transient paresis or paralysis is observed. With TIA in the vertebral-basilar pool, the main clinical symptom is dizziness.

The purpose of our work was to study method of ultrasonic Doppler examination and computer tomography for TIA diagnosis. The study included 76 patients (47 men and 29 women, aged from 45 to 80 years (mean age 62.3 ± 5.1 years) Control group (15 patients) included practically healthy individuals of the same age, with no signs of cerebrovascular insufficiency.

TIA etiological factors in our observations were as follows: cerebral atherosclerosis 68.42% (52 patients) and a combination of cerebral atherosclerosis and hypertension 31.57% (24 patients). The course of hypertension in our observations was malignant with average indices of blood pressure of 170 ± 6.4 mm Hg and frequent increases in systolic blood pressure over 220 mm Hg, worsening the picture of cerebrovascular insufficiency.

In the clinical picture, outside TIA, cerebral complaints to headaches, dizziness, which intensified at head turn and change of the body position, sleep disturbances, performance impairment and memory problems prevailed. In the presence of diffuse focal symptoms in the form of tendon anisoreflexia, oral automatism symptoms, pathological stop signs, most of our patients (80.2%) were diagnosed with dyscirculatory encephalopathy. It should be noted,that in most patients, clinical symptoms spoke for dyscirculatory encephalopathy, primarily, in the vertebral-basilar pool. In the latter case, there was vertebral-basilar insufficiency of II or III degree, which was also determined by the method of ultrasonic Doppler examination and computer tomography. The study of cerebral hemodynamics and main artery of the head was performed using ultrasonic Doppler examination on VAZOSKAN device, manufactured by Sonicaid (England) using sensors with

a frequency of 2.4.8 MHz and diameters of 14, 10 and 6 mm. The studies were performed in the city diagnostic centre.

According to Spiridonov A.A. (1996), pathology of only one carotid artery occurs in 51.7% of cases causes TIA development. The lesion of one vertebral artery was observed in 38% of cases. The incidence of multiple BCA lesions ranges from 50 to 93.8%. To determine the role of occlusive lesions in TIA development, ultrasonic Doppler examination was performed. The study of cerebral hemodynamics and main artery of the head was performed using ultrasonic Doppler examination on VAZOSKAN device, manufactured by Sonicaid (England) using sensors with a frequency of 2.4.8 MHz and diameters of 14, 10 and 6 mm.

Magnetic resonance imaging studies were performed in Radiology Department of the 3rd clinic of Tashkent Medical Academy using Magnetom Open Viva (Siemens) device with a magnetic field strength of 0.2 Tesla in coronary and transverse projections using a common flexible RF coil for the patient in a neutral position on his back.

Results

The patients' condition upon admission in most cases 80% (32 patients) was regarded by us as moderate. It is important to note that in most cases, the severity of patient's condition was due to a number of neurological symptoms, but not impairment of consciousness. For description of transient disorders, the following aspects were evaluated: TIA duration and incidence, probable vascular pool, morbidity, prevalence of focal or cerebral symptoms. Moreover, in most of our observations, TIA were predominantly focal over cerebral. Anamnestic data of patients with TIA are shown in Fig. 1.

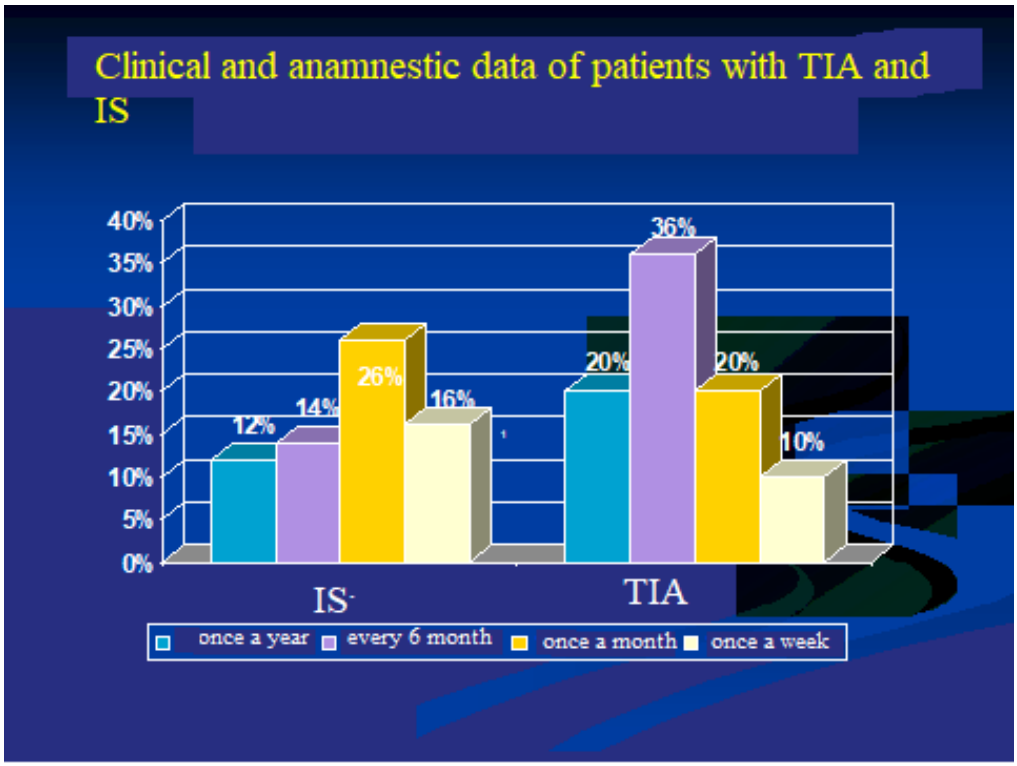


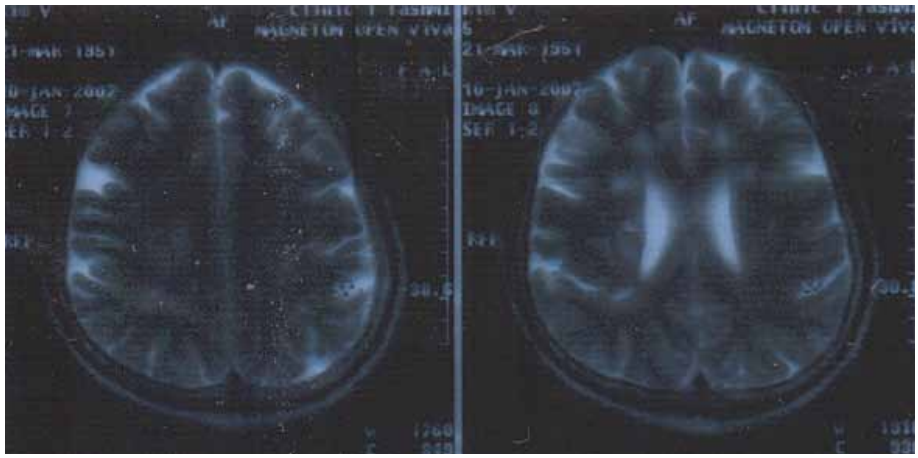
Figure 1. Clinical and anamnestic data of patients with TIA and ishemic stroke (IS).

As can be seen in Fig. 1, IS most often developed in patients with TIA incidence once a week or once a month. The highest risk of IS development was observed at a TIA incidence of once a week and reached 33.3%. The duration of TIA in this group of patients was up to 10-16 hours (according to anamnestic data). Based on clinical and anamnestic studies, we analysed the dependence of the risk of stroke development on the incidence of previous TIA in 18 patients who had a stroke against TIA (Fig. 2). In all cases, the stroke vascular pool coincided

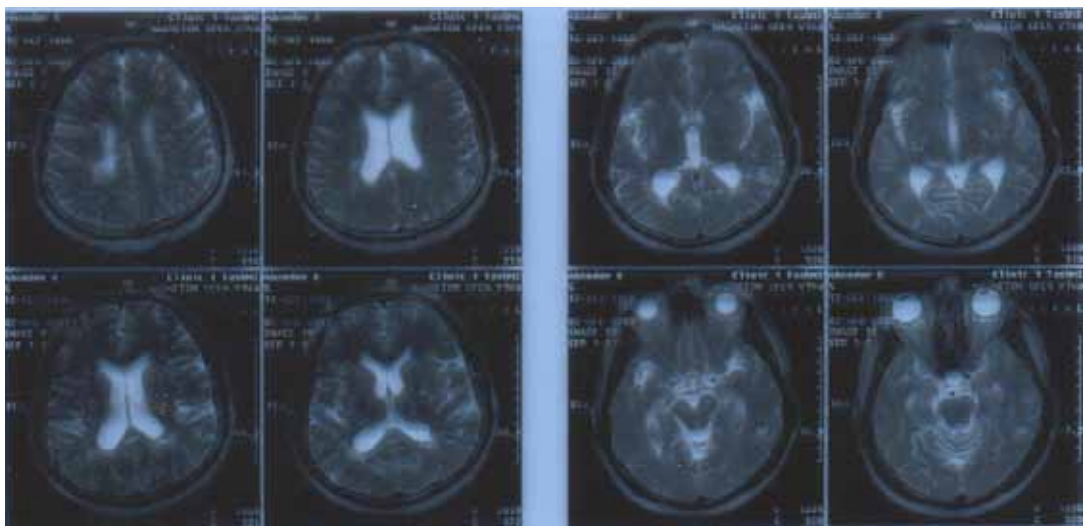
with the TIA localization. Besides, as presented data show, the incidence of stroke and TIA coincided when they were localized in the right carotid system (31% and 37%, respectively). At the same time, TIA in the left carotid system were observed 3 times less often (18%) than strokes in the same pool (55%). In contrast, TIA in vertebral-basilar pool were observed 3 times more often than strokes in the same vascular pool. The obtained results can be explained by anatomical features of the vascular pool structure, as well as by compensatory characteristics of these vascular pools.

The MRI studies revealed the dependence of TIA clinical picture the severity of brain atrophy. To determine the latter, based on MR images, we calculated indices of the lateral and third ventricles. Our studies showed that in the group of patients with frequent TIA (more than 2-3 times per month), indices of lateral ventricles were 1.3 and were much higher than in patients with TIA 1 time in 6 months or 1 year (0.8). Besides, dependence of MRI indices on the degree of dyscirculatory encephalopathy and etiological factors, caused cerebrovascular insufficiency.

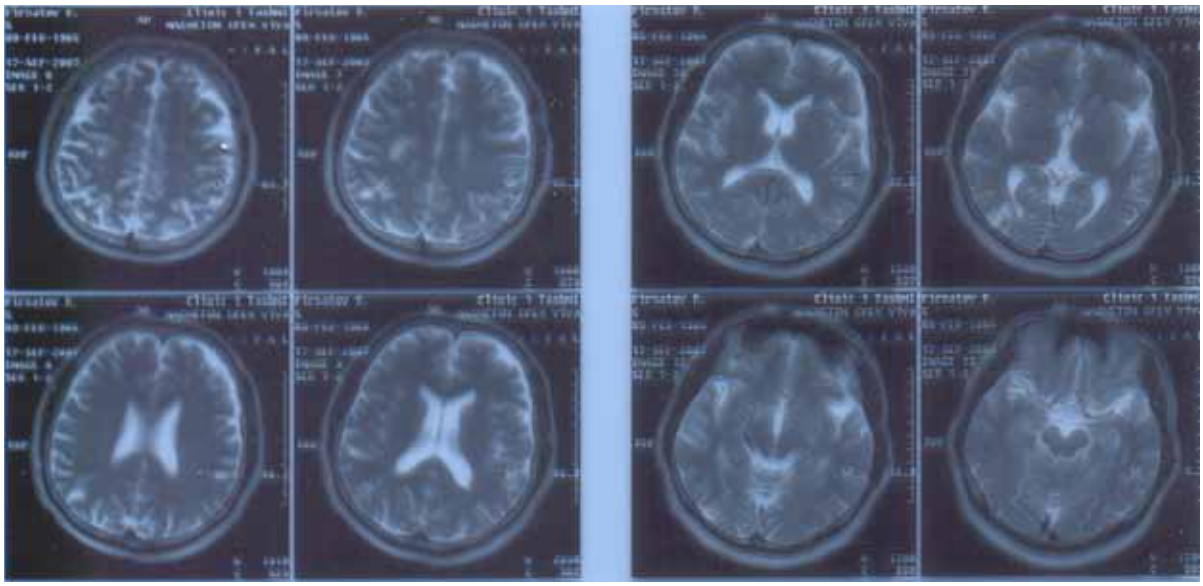
Multiple foci of reduced density no larger than 1 cm were found on MR images in the periventricular zone.



The number of the above foci also depended on the TIA incidence. Besides, in the group of patients with TIA incidence of more than 2-3 times a week, an expansion of subarachnoid space and sylvian fissures was observed, showing probable external brain atrophy and the state of cerebral hemodynamics. Typically, the cause of TIA in the latter case was a combination of arterial hypertension and cerebral atherosclerosis.



In patients with TIA on the background of cerebral atherosclerosis, we revealed only the expansion of sylvian fissures and subarachnoid space, with indices of lateral ventricles not significantly different from those of the control group.



Thus, as our studies showed, the clinical picture of TIA is characterized by various symptoms and diagnosis complexity, considering its retrospective character. The incidence, duration and vascular pool of TIA are correlated with the risk of cerebral stroke. The clinical picture of TIA is also largely determined by the brain condition, in particular, the severity of its atrophy. The latter, depending on the etiological factor, manifests itself in the form of predominantly external or internal forms. The number of foci of reduced density in the periventricular zone is also directly dependent on TIA incidence.

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Conflict of Interest: Nil

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