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Modern Approaches to the Diagnosis of Transistor Ischemic Attacks (TIA) (Review)

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Resume

currently, TIA is treated as a short episode of neurological dysfunction, which, as a rule, lasts less than 1 hour and has no symptoms of acute infarction, resulting from focal ischemia of the brain or clinical symptoms of the retina of the eye [7; 16]. This definition is generally well received and has been used in a number of stroke clinical trials (WARSS, RESPECT, etc. Proved by evidence in favor of a new definition of TIA.

Keywords: TIA, do' - MRT, TOAST.

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According to the 10th International Classification of globally accepted diseases (MKB10), transistor ischemic attacks are presented in the TIA "diseases of the nervous system" class under the heading "episodic and paroxysmal diseases", manifesting in itself sleep artery syndrome, temporary blindness, temporary global amnesia (code G45), damage syndromes of individual vascular pits of the brain (code G45) [2,3]. Generally accepted systems are used to classify ischemic strokes: TOAST (pathogenetic classification, including diseases of large or small vessels, embolism, other causes of stroke or unknown causes) [7], as well as modern SSS (Causative Classification System) and ASCO (Aphoratherosclerosis, Sforsmal lvesseldisease, Cforcardiacsource, Oforothercauses), systems are used to determine the underlying cause of stroke and reduce the number of uncertain etiology strokes with a combination of several causes [4; 13]. Although the cause of transistor ischemic attacks is considered an important predictor of the risk of further stroke, as found in some studies, there is little evidence of a need for TIA with an etiological classification [15; 16]. In a number of recent studies, the use of the TOAST, SSO, ASCO stroke subtype classification for transistor ischemic attacks has shown its value. M. In their work, Amort et al compared patients with diagnosed etiology of transistor ischemic ataka TIA, patients with unclear Tia etiology, and patients with etiological Tia subtypes with advanced or underdeveloped stroke within 3 months. The group of patients - 248 people (average age - 69 years, 60% of men), the duration of the Study - 2 years. The etiology of TIA has been determined by TOAST (35.9%), CCS (34.3%) and ASCO (38.7%). There was a significant difference between infestation classification systems for

unspecified causes of TIA, with this difference being the most minimal for ASCO (TOAST 46.4%; CCS 37.5%; ASCO 18.5%, R<0.001). Embolia (term 19,4/14,5/18,5%) and major arterial atherosclerosis (. 11,7/12,9/14,5%) were the most common causes of TIA according to TOAST, CCS and ASCO, respectively. After 3 months, recurrent ischemic stroke was observed in 33 patients (13.3%, 95% confidence interval II confidence index, 9.3-18.2%). Patients with studied transistor ischemic ataka Tia etiology had a higher risk of post-cranial circulatory abruption than patients of unknown cause: 4.4% (95% II, 2.5-14.1%) compared to TOAST 6.7% (95% II, 1.8-8.9%); CCS is 9.3% (95% II, 4.1-17.5%) compared to 3.1% (95% II, 1.0-7.1%); ASCO is 9.4% (95% II, 4.4-17.1%) compared to 2.6% (95% II, 0.7-6.6%). In patients with clearly confirmed Tia etiology, the risk of developing stroke compared to patients with unknown causes was clearly demonstrated in the Asso classification [4]. In contrast, another recent study found that J.A. Desai et al showed that among 469 patients with TIA or minor strokes, patients with multiple etiologies for CCS or ASCO or with unclear etiology for ASSO were more likely to experience a recurrent vascular damage event [8]. Nowadays, the Oxfordshire Community Stroke Project (OCSP) stroke classification system is popular, a system based on clinical syndromes that helps assess the risk of vascular damage events and recurrent stroke occurrence for TIA [6,9]. In accordance with this classification, the Centers corresponding to the carotid and vertebrobazillary basins of the cranial circulatory system can present in the anterior and posterior localizations of the cranial brain. As a result of the change in the concepts of the definition of transistor ischemic attacks over the past fifty years, there are 2 different definitions of TIA. In both cases, these definitions are described as a short episode of neurological dysfunction as a result of focal ischemia of the cranial or cornea, which is not associated with the formation of a cranial infarction [7]. Previously, TIA was presented as a sudden onset of pathological symptoms in a particular socket of the brain that last less than 24 hours, namely neurological deficits limited by the circulatory circle of one artery. Typical symptoms of TIA are: hemiparesis, hemigipesthesia, dysarthria, aphasia, diplopia, Association in the perioral zone, impaired movement coordination Si, and monocular blindness [1,6,8]. The twenty-four hour marking system was used from the mid-1960s to distinguish Tia from stroke. At that time, it is believed that during this period, symptoms disappear completely due to the absence of damage to brain tissue. At this time, another category was proposed, which was defined as a neurological deficit that recurs over a period of 24 hours to 7 days. Only symptoms lasting more than 7 days were assessed as cerebral infarction and thus the definition of "stroke" was established. In the 1970s, studies emerged confirming that most strokes lasting 24 hours to 7 days were associated with infarction, confirming that the term "reversible ischemic neurological deficit" was incorrect, and it was excluded from standard nomenclature [4]. In 1958, the U.S. National Institutes of health accepted Fisher's suggestion that TIA could last for several hours, but typically the duration of symptoms ranged from a few seconds to 10 minutes. In 1964, Acheson and Hutchinson state that the maximum duration of symptoms to distinguish TIA from stroke is one hour. In addition, in 2000-2020, Marshall proposed to accept the maximum duration of symptoms as 24 hours, but according to his data, the duration of siptoms lasted almost 1 hour when most patients were controlled. The 24-hour limit for TIA was adopted during a discussion at the IV Princeton blood conference in 1965 and was included in the 1975 U.S. National Institutes of Health classification. Between the 80s of the last century, computed tomography (CT) and diffusion-dimensional magnetic resonance tomography (do'-MRI) showed that most ischemic episodes lasting less than 24 hours (a third of TIA according to the literature) were associated with the formation of new infarcts [4]. These data highlighted the discrepancy between the concept of TIA (ischemia causes symptoms but does not damage brain tissue) and the original definition of TIA. With these observations, a group of scientists proposed a different definition of TIA in 2002. Currently, TIA is treated as a short episode of neurological dysfunction, which, as a rule, lasts less than 1 hour and has no signs of acute infarction, resulting from focal ischemia of the brain or clinical symptoms of the retina of the eye [7; 16]. This definition is generally well received and has been used in a number of stroke clinical trials (WARSS, RESPECT, etc. Proved by evidence in favor of a new definition of TIA:

1.The classic 24-hour limit for TIA has been perceived as incorrect, as in many patients with reversible

symptoms, brain tissue damage has developed in this time frame, which has been confirmed by a number of studies [16, 19; 18]. Thus, the 24-hour duration of symptoms cannot clearly distinguish between patients with and without cerebral infarction (Category III, evidence level A). S.H.Observations from Shah et al have yielded results showing the duration time of Tia characters and the presence of high density flies in the cranium according to DV-MRT [5,8].

2. . Thrombolysis in ischemic stroke should be done as early as possible, while the method of detecting 24-hour Tia can incorrectly stimulate waiting tactics.

3. Many studies show that TIA usually lasts less than 1 hour. When analyzing the results of magnetic resonance imaging (MRI) of patients with TIA, 60% of the events lasted less than 1 hour, 71% lasted less than 2 hours, and only 14% lasted more than 6 hours. Damage to the cranial tissue does not depend on the duration of the symptoms and in this case cannot be decisive even for the diagnosis of 24-hour TIA.

4. The fact of ischemic damage to the tissues of the same organ is used in the diagnosis of diseases of other organs. For example, stenocardia differs from myocardial infarction not by the duration of the disease, but by signs of damage to the myocardial tissue. The definition of TIA based on the duration of symptoms in this case can lead to distraction from the pathophysiology of the process.

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