Transient ischemic attack in the vertebrobasilar vascular territory as a cause of isolated vertigo

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Transient ischemic attack (TIA) in the vertebrobasilar vascular territory (VB) causes difficulties in diagnosis when it manifests only with vestibular symptoms. Issues relating to the differential diagnosis of TIA are discussed, awareness of which is necessary for the selection of informative methods of examination and the prescription of effective stroke prevention in patients with an episode of isolated dizziness. The likelihood of TIA as the cause of dizziness is increased by the patients' high cardiovascular risk, the presence of atrial fibrillation, severe instability during an attack, and head and/or neck pain. If a TIA in VB is suspected, it is advisable to perform a minimal instrumental examination, including computed tomography (CT) of the brain and CT angiography or diffusion-weighted magnetic resonance imaging (MRI) and MRI angiography. In case of doubt, additional information can be obtained by a perfusion CT or MRI as well as a post-contrast MRI. When interpreting the results of these methods of examination, their limitations in terms of application time and resolution should be taken into account.

Keywords: *transient ischemic attack in the vertebrobasilar vascular territory; diagnosis; differential diagnosis; CT scan; magnetic resonance imaging.*
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Differential diagnosis of acute dizziness is well covered in the scientific literature and is actively being introduced into clinical practice [1P5]. Work on this issue is predominantly focused on Meniere's disease [6], vestibular migraine [7], persistent postural perceptual vertigo [8] and, as a rule, does not address the issues of isolated dizziness in transient ischemic attack (TIA). At the same time, throughout the world, TIA in posterior circulation (PC-TIA) causes difficulties in diagnosis if it manifests itself only with vestibular symptoms. In real practice, the following features can be identified in relation to acute vestibular syndrome: 1) poor knowledge of vestibular migraine and peripheral vestibular disorders; 2) the difficulty of coding these diseases in departments for patients with stroke; 3) higher cost of a stroke case, reducing the motivation to code the disease as migraine or peripheral vestibulopathy. In this lecture, we tried to highlight the most difficult positions in the differential diagnosis of TIA, knowledge of which is necessary for choosing informative research methods and prescribing effective stroke prevention in patients with an episode of isolated dizziness.

Definition

According to the definition American Heart Association / American Stroke Association (AHA/ASA) 2023, TIA is an acute neurovascular syndrome related to a specific arterial territory, which quickly regresses, leaving no signs of infarction on the diffusion weighted image (DWI) of magnetic resonance imaging (MRI) [9]. In contrast to the previously accepted definition, based on the time parameter (24 hours), the modern definition reflects the priority of the TtissueУ factor, therefore, in the presence of a small infarction on DWI, even in the case of regression of symptoms within a few minutes, the diagnosis of TIA is changed to ischemic stroke [10, 11].

In recent years, the risk of stroke in patients with TIA has decreased, due to the emergence of more effective prevention [12]. Thus, in the international TIA registry (analysis for 2016), the risk of IS after TIA or minor stroke was 1.5% within 2 days, 2.1% in the first 7 days, 2.8% in the first month, 3.7 % in the first 3 months and 5.1% in the first year [13]. However, these encouraging data cannot be transferred to all patients with TIA: in the presence of stenosis of the arteries of the intracranial artery, the risk of stroke within 3 months reaches 25% and is maximum (33%) in atherosclerosis of the intracranial arteries [14]. Given that atherosclerotic disease of the cerebral arteries occurs in at least 12% of the European population [15] and in almost half of people aged 90 years and older [16], any PC-TIA should be considered a high-risk cerebral vascular event. The problem is compounded by the fact that PC-TIA are difficult to diagnose. Thus, the Oxford Vascular Study showed that TIA are 15 times more likely to precede vertebrobasilar than carotid stroke, especially during the last two days (the risk is 36 times higher), and are often presented with isolated dizziness [17]. Given these data, the main emphasis in this lecture will be on the differential diagnosis of PC-TIA. But first we should discuss the clinical manifestations of classic TIA.

Spectrum of clinical manifestations of TIA

Symptoms of TIA usually last from a few seconds to several minutes and in typical cases resolve within one hour [11]. The most characteristic manifestations of TIA are monocular blindness, hemiparesis (two of the three body parts – arm, leg or face), aphasia, dysarthria and homonymous hemianopsia, which develop suddenly [18, 19]. It is noteworthy that the sudden development of symptoms, their duration of more than 1 minute, as well as age 60 years and older, increase the likelihood of TIA in women, and unilateral sensory loss and pain in men [19]. In addition to the classic symptoms of TIA, there are atypical manifestations, which mostly relate to PC-TIA (see table). The Oxford Vascular Study demonstrated that atypical TIA do not differ from classic attacks in terms of short- and long-term risk of stroke [20].

Symptoms suggestive of a disorder other than TIA include atypical isolated phenomena such as amnesia, confusion, incoordination of limbs, partial sensory deficits (unusual sensations or deficits limited to one limb or only the face), unusual cortical visual phenomena (positive symptoms – flashes, stars, colored dots, curls, as well as distortion, tilting of images, visual trail, hallucinations), loss of consciousness and headache [11]. History data that cast doubt on TIA include the patientXs young age in the absence of vascular risk factors (the exception is TIA due to the mechanism of paradoxical embolism, against the background of dissection or reversible cerebral vasoconstriction syndrome), epilepsy, migraine without aura (may be associated with dissection) , migraine with aura (may be associated with patent foramen ovale) [9]. Next, we will discuss isolated dizziness, as the most common situation requiring differential diagnosis of TIA with other conditions.

Symptoms of TIA in the VB that cause difficulties in diagnosis

Symptom	Description	Differential diagnosis
Isolated dizziness	Spontaneous dizziness (possible nausea and/or vomiting) without tinnitus, hearing loss or pain; does not include nonspecific dizziness and a feeling of lightheadedness	Vestibular migraine, Meniere's disease, benign paroxysmal positional vertigo, persistent postural perceptual vertigo
Isolated ataxia	Unsteadiness when walking	Vestibular migraine, migraine with brainstem aura, episodic ataxia, epileptic pseudoataxia, paroxysmal dyskinesia, functional neurological disorder
Isolated diplopia	Binocular diplopia	Myasthenia gravis, migraine with brainstem aura, ophthalmoplegic migraine, thyroid ophthalmopathy
Isolated dysarthria	Slurred speech	Myasthenia gravis, migraine with brainstem aura
Bilateral vision loss	Hemianopsia or quadrantanopsia without positive visual phenomena	Migraine, posterior reversible encephalopathy syndrome, reversible cerebral vasoconstriction syndrome

Isolated dizziness

In 12% of patients with PC stroke, transient vestibular symptoms are observed within 3 months (in a third – within a week) before the disease, in $^2/_3$ of cases without imbalance [21]. It is noteworthy that in half of the patients, dizziness increases with changes in head position, which creates a risk of misdiagnosis of benign paroxysmal positional vertigo. In half of the patients, dizziness lasts only a few seconds, in a third it lasts minutes; in $^3/_4$ of patients these episodes are repeated 1 to 5 times [21]. On the other hand, the results of the study by A.K. Bery et al. [22] indicate a low risk of ischemic stroke after an episode of isolated dizziness – less than 1% within 3 months. Thus, transient dizziness is a very heterogeneous condition both in terms of etiology and prognosis.

What is recurrent vestibular syndrome? The syndromic diagnosis of transient isolated dizziness is associated with the concept of episodic vestibular syndrome, which includes vestibular migraine and MenіereXs disease (characterized by recurrence over many years) as the main TbenignУ causes of spontaneous dizziness, and TIA as dangerous ones [23].

Despite detailed otoneurological examination and neuroimaging (including DWI and perfusion techniques), the etiology of more than half of cases of transient isolated vestibular vertigo (TIVV) remains unknown [24].

How common is isolated vascular vertigo? Isolated dizziness is the only clinical manifestation in every fifth patient with PC-TIA [25]. Stroke occurs in 27% of patients visiting the emergency department for transient vestibular syndrome [24]. According to F. Nikles et al. (Switzerland) [26], stroke is diagnosed in 13% of patients in the emergency department with acute development of vestibular symptoms, while every 20 of these patients previously had vestibular syndrome. In a study by J.H. Choi et al. (South Korea) [24] among patients with the first attack of dizziness in their life, 27% were diagnosed with a stroke. In a large study conducted in Switzerland (2023), among patients who visited the emergency department with transient dizziness, stroke was diagnosed in 2%, TIA in 10%; in almost half of the cases, the etiology of dizziness remained unknown [27].

Damage to which parts of the brain is associated with transient vascular vertigo? Cerebellar infarction accounts for 10 of 13 cases of stroke with TIVV [24]. A short duration of symptoms is characteristic of the localization of the infarction in the lateral and caudal parts of the cerebellum (posterior inferior cerebellar artery) and the parietoinsular cortex [28]. It is important to note that isolated vestibular symptoms are observed in 1% of hemispheric stroke cases [29] and are usually associated with damage to the vestibular cortex of the right hemisphere [30].

How to clinically suspect TIA in a patient with dizziness? Dizziness with PC-TIA is usually spontaneous, lasts several minutes and does not have any reliable distinguishing features [25].

Anamnesis

The presence of cardiovascular risk factors in a patient (old age, arterial hypertension, atherosclerosis, coronary heart disease, atrial fibrillation, diabetes mellitus, history of stroke) increases the likelihood of vascular genesis of TIA, but should not be considered as a leading criterion. A significant argument in favor of TIA is the absence of a history of dizziness [5, 31].

The presence of neck pain and headache increases the likelihood of stroke in TIVV by 15 times [24]. In acute vestibular syndrome, moderate or severe truncal ataxia is observed only with stroke [5, 32], as well as the inability to sit (sitting ataxia) [33].

Unfortunately, the use of the HINTS protocol, which is an ideal tool for differentiating central and peripheral acute vestibular syndrome, is not informative for TIVV, since patients do not have symptoms at the time of visiting a doctor [5, 24].

Instrumental diagnostic methods

Their use helps solve two problems: visualization of the focus of cerebral infarction and verification of diseases that could lead to transient vascular vertigo.

With regard to the first task, it should be noted that non-contrast computed tomography (CT) of the brain is practically uninformative in differentiating the causes of TIVV [5, 9]. Thus, in patients with dizziness of unknown etiology (without typical signs of peripheral dizziness and focal neurological deficit) who come to the emergency department, CT of the brain is diagnostically useful in only one case out of 100 [34].

MRI (DWI) allows visualization of acute infarction (cortical) in approximately half of patients with TIA [35, 36].

The likelihood of DWI positivity is higher in patients with cardioembolic TIA [37], the presence of hemiparesis [38] and lower in young patients [39].

Infarction on subsequent MRI is detected in every fifth initially DWI-negative patient. Moreover, every fifth new lesion is located in the brainstem [40]. It is noteworthy that the probability of visualizing an infarction is the lowest in the first 12 hours after TIA (8.7%), while beyond the first 24 hours, within 2 weeks, it is about 30% [41]. When performing primary DWI in the first 2 hours from the development of TIA symptoms, repeated imaging is necessary to exclude a false negative result, which will be observed in every 4P5 patients [42].

Also, when analyzing MRI, attention should be paid to the presence of vascular hyperintensity on FLAIR images: this phenomenon is associated with delayed appearance of the lesion on DWI [43].

In the absence of a lesion on DWI, neuroimaging can be expanded. Thus, the addition of a contrast agent to MRI may allow visualization of the hyperintense acute reperfusion marker (HARM), which is observed in every tenth patient with transient neurological symptoms. At the same time, penetration of gadolinium into the anterior chamber of the eye (gadolinium leakage in ocular structures, GLOS) is observed three times more often [44].

Another option for evaluating DWI-negative patients may be perfusion-weighted MRI [45]. In 12% of patients with PC-TIA, unilateral cerebellar hypoperfusion without infarction is detected according to perfusion-weighted MRI, and most of them have stenosis or hypoplasia of the corresponding vertebral artery (VA) [24]. In a study by D.P. Zhang et al. (China) [46] showed the high value of visual assessment of MRI perfusion for the diagnosis of isolated vascular vertigo.

Based on an analysis of errors in diagnosis, L. Comolli et al. [27] recommend delayed neuroimaging for TIVV.

Solving the second problem (searching for a cardiovascular cause of dizziness) involves, first of all, performing CT or MRI angiography. Presence of VA stenosis (usually in segment V4) or basilar artery (BA) in a middle-aged or elderly patient will indicate a vascular origin of dizziness. Detection of stenosis or hypoplasia of the VA increases the likelihood of stroke in transient vestibular syndrome by 7 times [24].

Also, CT angiography allows visualizing signs of dissection [47]. In addition, if the patient has cardiovascular risk factors, it is necessary to perform electrocardiography, Holter monitoring and echocardiography.

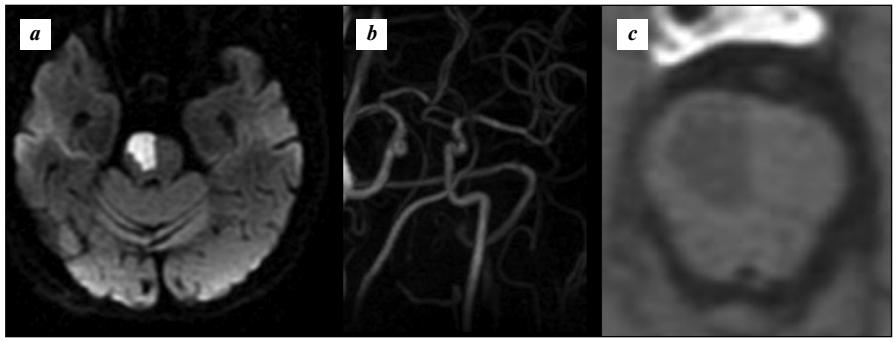

Fig. 1. *Clinical example of a TIA in the VB.*
A female patient, 74 years old, has a long history of arterial hypertension and type 2 diabetes mellitus. At 3:00 a.m., she went to the bathroom, felt severe dizziness, fell and could not get up for an hour. An hour later, the dizziness disappeared. In the morning she called an ambulance and was admitted to the primary vascular department. A CT scan of the brain was performed, which revealed no signs of a stroke. As there were no complaints at the time of the examination, no focal neurological deficit was detected and the dizziness was categorised as non-specific, the patient was discharged. In the evening of the same day, she developed weakness in her left limbs, facial asymmetry, double vision and slurred speech; there was no dizziness recurrence. Twelve hours after the onset of symptoms, she was admitted to the emergency department and hospitalized. On admission, internuclear ophthalmoplegia, nuclear prosoparesis, left-sided hemiparesis and hemihypaesthesia were diagnosed. An MRI of the brain was performed and a right paramedian pontine infarction was detected (a, DWI-MRI). At the level of the infarction, a stenosis of the basilar artery of about 50 % was detected (б, MRI INHANCE) with contrast enhancement in the plaque (в, MRI, T1). Dual antiplatelet therapy with acetylsalicylic acid and ticagrelor was prescribed. There was no progression of the neurological deficit during treatment. After 10 days, the patient was able to walk with aid and was referred to the medical rehabilitation department

Diagnostic criteria

Diagnostic criteria for probable transient vascular vertigo were proposed by the Barany Society in 2022.

Diagnostic criteria for probable transient vascular vertigo [5, 25]

A. Acute spontaneous dizziness or unsteadiness lasting less than 24 hours

B. At least one of the following criteria:

1. Focal central neurological symptoms or severe postural instability during the attack.
2. New-onset craniocervical pain of moderate or severe intensity.
3. Increased risk of vascular complications (for example, ABCD2 scale score 4 or more or atrial fibrillation).
4. Significant (>50%) narrowing (hypoplasia/stenosis) of the artery in the vertebrobasilar system

B. Other diseases that could cause the corresponding symptoms have been excluded

A clinical example of PC-TIA is presented in Fig. 1.

Diagnostic algorithm

A possible differential diagnosis algorithm for the first episode of isolated dizziness in life is presented in Fig. 2.

Secondary prevention

Secondary prevention for TIA consists of control of risk factors, antithrombotic, lipid-lowering and antihypertensive therapy, as well as surgical methods of prevention. In case of TIA against the background of known atrial fibrillation, immediate administration of a direct oral anticoagulant is advisable [9, 48]. Approaches to secondary prevention of high-risk non-cardioembolic attacks include short-term dual antiplatelet therapy, long-term dual antithrombotic therapy with acetylsalicylic acid and low doses of rivaroxaban (for multifocal atherosclerosis and low hemorrhagic risk), urgent revascularization (for carotid TIAs) and intensive, including combined , lipid-lowering therapy [49]. When choosing a combination of drugs for dual therapy, it is advisable to focus on the result of the ABCD2 scale: with a value of 4P5 points, a combination of acetylsalicylic acid and clopidogrel is indicated, with a value of 6 points and above, acetylsalicylic acid and ticagrelor are indicated [9, 49]. In our opinion, due to the extremely high risk of stroke in patients with atherothrombotic PC-TIA (especially against the background of atherosclerosis of the intracranial arteries), the use of ticagrelor is preferable in this situation [50].

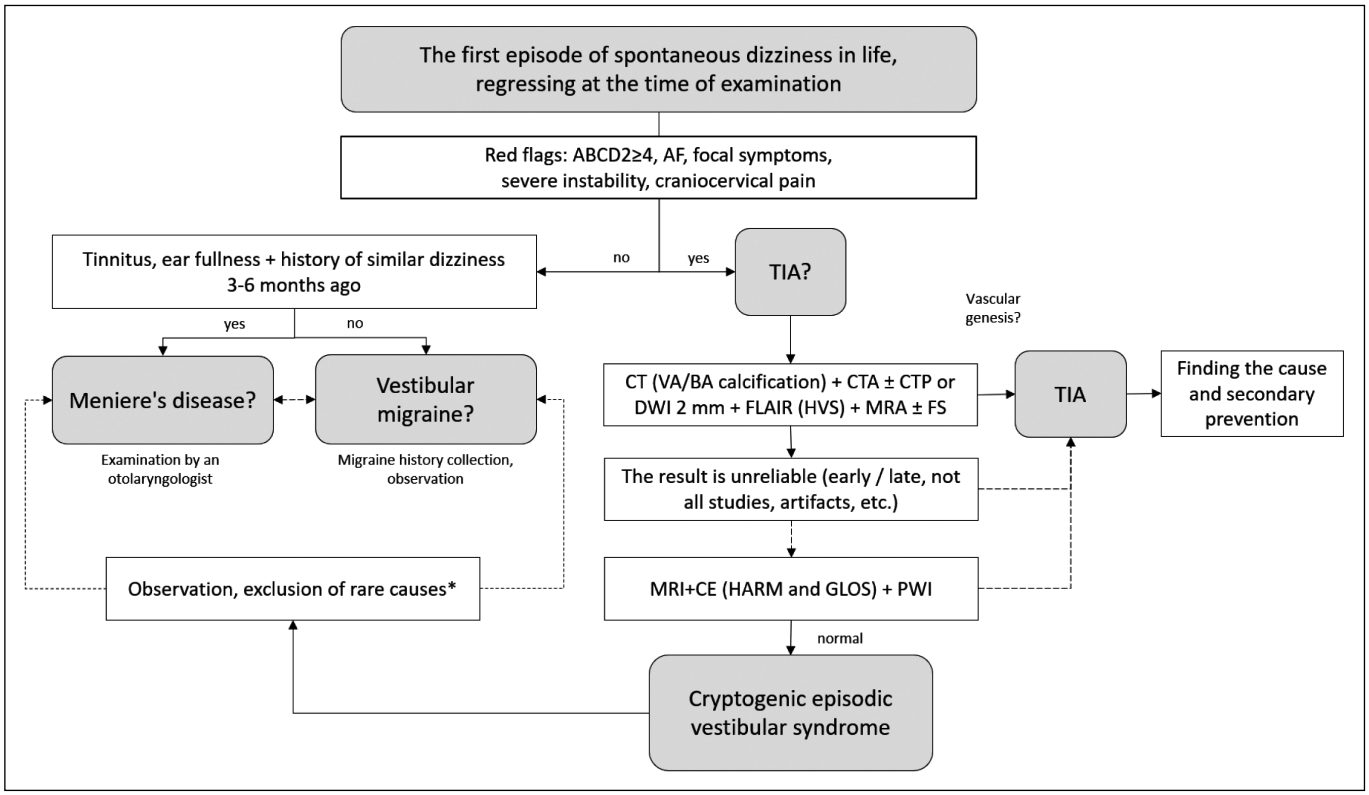

Fig. 2. *Differential diagnosis algorithm for the first in life episode of spontaneous dizziness that has resolved at the time of examination. * – vestibular paroxysmia, third-window syndrome (dehiscence of the anterior, posterior and lateral semicircular canal), vestibular schwannoma, labyrinthine schwannoma, dilatation of the vestibular aqueduct*

Conclusion

Isolated dizziness can serve as a manifestation of PC-TIA. The likelihood of a TIA as a cause of dizziness is increased by the patient's high cardiovascular risk (assessed by the ABCD2 scale), the presence of atrial fibrillation, severe instability during an attack, as well as headache and/or neck pain. If a PC-TIA is suspected, it is advisable to perform a minimal instrumental examination, including CT of the brain (focus on calcifications in the VA and BA) and CT angiography or DWI MRI and MRI angiography. In doubtful situations, additional information can be obtained using perfusion CT or MRI, as well as post-contrast MRI. When interpreting the results of these research methods, one should take into account their limitations associated with the period of application and resolution.

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