IJCNP, Volume 2, Issue 9, 2024 ISSN: 2995-536X
https://medicaljournals.eu/index.php/IJCNP

INTERNATIONAL JOURNAL OF COGNITIVE NEUROSCIENCE AND PSYCHOLOGY

THE IMPORTANCE OF AN INTEGRATED CLINICAL AND NEUROIMAGING APPROACH IN THE CORRECT DIAGNOSIS OF VASCULAR VERTIGO

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Abstract: The article provides information devoted to modern ideas about terminology, etiology, pathogenesis, clinical features and diagnosis of vertigo of vascular etiology. The variants of vestibular disorders in ten different acute and chronic diseases related to cerebrovascular pathology are analyzed. The importance of complex clinical and neuroimaging approach in the correct diagnosis of vascular vertigo is emphasized. The most frequent errors and misconceptions of neurologists with regard to vertigo caused by ischemic and hemorrhagic brain diseases are summarized. Key words: vestibular syndrome, acute and chronic cerebrovascular pathology.

Introduction. Vertigo is one of the frequent and sometimes the only complaint of patients seeking medical help. Despite its apparent simplicity, its diagnosis and treatment often cause great difficulties. At the same time, some neurologists make primitive mistakes or have common misconceptions when evaluating dizziness. The first mistake is often the misunderstanding by physicians of the term “vertigo” as a basic part of vestibular syndrome (VS) or vertigo. It is generally accepted that VS refers to the sensation of imaginary movement of objects or one's own body in space or distortion of gravitational orientation [2, 10]. In practice, some specialists sometimes mistakenly understand VS as patients' complaints of non-vestibular symptoms, such as general weakness, malaise, malaise, malaise, anxiety, pre-syncope, and so on. The reason for this misconception often lies in the fact that the HC patient does not usually share their complaints when seeking help. In this regard, it is worth paying attention to the fact that VS includes three groups of symptoms: 1) nystagmus or involuntary eyeball movements with many characteristics; 2) static and gait disorders; 3) various autonomic and vestibulovigilance disorders [3, 7, 9-11]. It is important to remember that VS is classified into central and peripheral, in which these manifestations occur in different severity and combinations, and is also divided into acute and chronic [7, 13]. The second characteristic misconception of neurologists in Belarus is overdiagnosis of vascular genesis of VS, especially in elderly and old people. Meanwhile, according to the results of epidemiologic studies, there are many known causes of VS: metabolic, toxic, inflammatory, traumatic, psychiatric, hereditary, etc. The vascular etiology is the most common cause of VS. At the same time, vascular etiology in the structure of VS in general makes up only 12.2% [13]. However, even despite the relative rarity of the vascular genesis of vertigo, it should not be forgotten, as the erroneous diagnosis of this pathology is fraught with unfavorable consequences for patients, including lethal outcomes.

Three groups of diseases related to cerebrovascular pathology can be distinguished:

I. VS caused by acute ischemia in the basin of the vertebrobasilar artery system (VBBA):

1) isolated infarcts of medulla oblongata and pontine;

2) isolated cerebellar infarcts;

3) cerebral infarcts in the basin of the anterior inferior cerebellar artery;

4) cerebral infarcts in the posterior inferior cerebellar artery basin;

5) transient ischemic attacks (TIAs) in the VBBA;

6) labyrinthine infarcts;

7) vertebral artery syndrome.

II. VS due to chronic cerebral ischemia:

1) initial manifestations of cerebrovascular insufficiency;

2) dyscirculatory encephalopathy of I-III degree.

III. VS caused by hemorrhage in the brainstem or cerebellum.

In patients with all the above diseases, vascular dizziness occurs as part of the clinical picture. However, the sun in each of them has its own characteristic features, knowledge of which in some cases significantly facilitates its diagnosis. The causes of vascular BC are more often arterial hypertension, obliterating atherosclerosis of the brachiocephalic arteries and diabetes mellitus. Other etiological factors, such as cardioembolism, arterial dissection, bone or vascular abnormalities, degenerative-dystrophic changes in the cervical region, are observed much less frequently. Hypoxia plays a leading role in the pathogenesis of vascular vertigo, since the labyrinth is most susceptible even to short-term hypoperfusion, which always occurs for any of these reasons [2, 6].

With all these nosological forms, BC can be central, peripheral, or mixed, which gives it a pronounced clinical polymorphism that determines diagnostic errors. In this regard, it is necessary to highlight the third misconception of neurologists in our country (however, it is typical for the entire post-Soviet space) that the main role in the recognition of these diseases is played by neuroimaging methods, in particular CT or MRI of the brain. Without detracting from the importance of a competent assessment of radiation methods, it is worth recalling that in practice there are false positive (confirming the diagnosis of stroke) and false negative (excluding) results. Therefore, knowledge of the anamnesis, clinical features and objective manifestations of VS in the diagnosis of this pathology can be crucial. In addition, the diagnosis of vascular etiology of VS must be confirmed by the results of instrumental studies not only of the brain, but also of the extracranial department of the main arteries (especially during their dissection).

We will briefly present the main clinical and neuroimaging characteristics of diseases accompanied by the vascular genesis of VS.

Isolated infarctions of the medulla oblongata and bridge

This localization is very rare (1%) and is characterized by a variety (more than 13 variants) of well-known neurological syndromes with eponymous names by the last names of the authors who first described them. It should be borne in mind that in clinical practice, classical alternating syndromes are practically not found (usually in different combinations), with the exception of Wallenberg - Zakharchenko syndrome (lateral medullary infarction). It usually develops with damage to the vertebral artery and is characterized, in addition to acute dizziness, by heterolateral hemianesthesia, hemiparesis, or hemiataxia dysphagia, ipsilateral Jurner syndrome [3, 11]. Objective signs of SUN in this case, as a rule, are multiple nystagmus in two or more directions; vertical deviation of the eyeballs when fixing the gaze (deviation of the eyeball on the side of the lesion down and inwards, the other - up and outwards) and the head (tilted towards the focus); dizziness is not systemic, nausea and vomiting are not expressed. With medial localization of the infarction in the area of the bridge, the SUN is complemented by symptoms of damage to the abductor, facial or oculomotor nerves on the side of the infarction [2, 10]. Confirmation of the diagnosis is the detection of a single lacunar focus of reduced density in the brain stem or bridge area during CT scan (Fig. 1).

Isolated cerebellar infarction

Cerebral hemisphere and (or) cerebellar worm infarction occurs more often and is always accompanied by acutely developed (usually against the background of decompensation of arterial hypertension) VS. Its typical features are spontaneous and only horizontal nystagmus, pronounced imbalance with a fall towards the lesion, and blurred vegetative symptoms. In such patients, in addition, the neurological status determines a decrease in muscle tone in the opposite extremities and chanted speech [6]. The fundamental difference between this cause of BC development and any peripheral BC is the absence of

changes during caloric tests [2]. When conducting a CT scan of the brain, it is typical to identify the focus of a heart attack of this localization.

Cerebral infarction in the anterior inferior cerebellar artery basin

An isolated infarction in the blood supply basin of this artery is a rarity in angioeducology. Clinically, a cerebral infarction of this localization in 98% of cases is manifested by acute VS with the classical triad of symptoms described above [9]. Neurological disorders that allow the clinician to suspect this pathology are that dizziness in a patient with a brain infarction in this case may be of a mixed nature - central and peripheral (65% of cases) or only central (33% of cases) [2]. For this reason, acute systemic and (or) non-systemic dizziness and horizontal (or vertical) spontaneous nystagmus are sometimes combined with hearing loss or noise in the ear and involvement of the facial and abductor cranial nerves [10]. In this case, a unilateral decrease in vestibular excitability during a caloric test, confirming | CT scan of the brain: Varoliev bridge infarction on the left, damage to the labyrinth, may lead to a diagnostic error. As a result, due to echeloned thrombosis or progressive dissection, not only a heart attack can develop in the anterior-inferior parts of the bridge and the middle pedicle of the cerebellum (the area of arterial blood supply), but also an extensive infarction in one occipital lobe. Cerebral infarction in the basin of the posterior inferior cerebellar artery

In patients with cerebral infarction in the blood supply basin of this artery, as in the previous case, acutely developed BC can have both a central and mixed (central and peripheral) character. In this regard, patients' complaints of systemic (rotational) and non-systemic (feeling intoxicated) dizziness may be accompanied by multiple nystagmus, nausea and vomiting, shakiness, dysmetria, as well as unilateral hearing loss [7]. A feature of the neurological status for a heart attack of this localization is nystagmus, which changes its direction when the gaze changes (54% of cases), and pronounced torso ataxia [10]. An erroneous diagnosis of this infarction is dangerous due to subsequent swelling of the cerebellum and compression of the brain stem.

Transient ischemic attack in the VBBA basin

Currently, the most common definition of TIA is the sudden development of focal (limited by the area of blood supply to a separate vessel) cerebral neurological deficit that persists for no more than 24 hours [3, 6]. In routine neurological practice, we often find overdiagnosis of TIA in the BBBA pool even in specialized stroke departments, especially in elderly people with risk factors for cerebrovascular pathology. On the other hand, underestimation of this pathology has a higher risk of subsequent stroke than with TIA in the carotid basin [2]. Despite the fact that acutely developed transient non-systemic dizziness is the main manifestation of TIA in the VBBA basin, clinicians should remember that not always VS is a consequence of TIA. The fourth common neurological misconception is the establishment of almost always a diagnosis of TIA in the BBBA basin in patients with transient global amnesia (sudden short-term memory loss), which may have non-vascular genesis (epileptic, psychogenic, etc.). Symptoms that can confirm the diagnosis of TIA in the VBBA basin are simultaneously appearing visual disorders of various types (69%), attacks of sudden loss of consciousness with a fall and internuclear ophthalmoplegia (33%), dysarthria, mild vegetative disorders [7]. Repeated TIAs in the VBBA basin are characterized by the detection of critical vertebral artery stenosis during CT or MR angiography [11, 12].

Conclusions: Thus, dizziness is a polyethological clinical symptom. Vascular VS has various etiologies, numerous phenotypes, various neurological features, can be either isolated or occur in combination with other symptoms of damage to the nervous system. For the correct diagnosis of vertigo of vascular origin, a well-conducted neurological examination in combination with the results of neuroimaging is important. Timely diagnosis of vascular vertigo is the key to its successful therapy.

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