EXCELLENCIA
INTERNATIONAL MULTI-DISCIPLINARY JOURNAL OF EDUCATION
Volume 3, Issue 2, 2025 ISSN (E): 2994-9521

Specific Features of Dizziness Syndrome in Cerebrovascular Diseases

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Acute spontaneous vertigo can be a symptom of numerous and very diverse diseases. These diseases are united by one common sign - sudden damage to the vestibular system or its connections with other parts of the brain. Diseases leading to acute vertigo may affect the peripheral part of the vestibular analyzer (for example, the labyrinth of the inner ear or the vestibular nerve) or those components of the vestibular system that are located in the CNS, primarily in the brainstem, or have connections to the cerebellum. According to a study conducted in the United States [1], approximately 3-5% of visits to the doctor are caused by complaints of dizziness, i.e. 10 million people go to the doctor for dizziness in the United States every year. Of these, 25% go to the doctor for emergency indications in connection with an acute attack of dizziness. About 3% of emergency hospitalizations are due to dizziness. From a practical point of view, the most difficult is the differential diagnosis of acute vestibular vertigo for the first time, since recurrent and reversible vertigo in the overwhelming majority of cases is associated with more or less benign diseases of the peripheral part of the vestibular analyzer and is extremely rarely the result of repeated transient ischemic attacks or other diseases of the central nervous system. Among the most common causes of acute first-onset non-recurrent vertigo are vestibular neuronitis and stroke in the vertebrobasilar system. Vestibular neuronitis is associated with selective inflammation of the vestibular nerve [2]. It is assumed that the cause of inflammation is the herpes simplex virus type 1 [3]. Vestibular neuronitis is manifested by a sudden and prolonged attack of systemic vertigo, accompanied by nausea, vomiting and imbalance. The disease may be preceded by a respiratory viral infection. Sometimes a few hours or days before the development of an acute vestibular attack, patients experience short-term episodes of dizziness or instability. Symptoms of vestibular neuronitis are aggravated by head movements or changes in body position, but unlike benign paroxysmal positional vertigo, they do not go away at rest. Dizziness may decrease when the gaze is fixed.Neurological examination shows no symptoms of damage to the brain stem or other parts of

the brain. Hearing in vestibular neuronitis does not decrease. In cases where sensorineural hearing loss develops simultaneously with dizziness, labyrinthitis is diagnosed [4]. The duration of vertigo ranges from several hours to several days. After the cessation of dizziness, patients continue to experience instability for several days or weeks. Then, due to the mechanisms of central vestibular compensation, the instability also regresses. Vestibular neuronitis recurs relatively rarely: according to various sources, in 1.9–10.7% of cases [5, 6]. Another, rarer, but much more formidable, cause of an attack of vestibular vertigo for the first time can be a stroke in the vertebrobasilar system. Stroke in the vertebrobasilar system accounts for about 20% of ischemic strokes, and one of the main manifestations of such a stroke is dizziness [7]. Dizziness during a stroke can be one of the symptoms of the disease or its only manifestation. In the first case, it is usually not difficult for a neurologist to diagnose a stroke. Among the infarctions in the vertebrobasilar system, accompanied by dizziness as one of the symptoms, the most common is infarction of the dorsolateral part of the medulla oblongata and the lower surface of the cerebellar hemisphere, which occurs due to occlusion of the vertebral or posterior inferior cerebellar artery. It is manifested by Wallenberg-Zakharchenko syndrome, which in the classic version includes dizziness, nausea, vomiting; on the side of the focus – pain and temperature hypoesthesia of the face, cerebellar ataxia, Horner's syndrome, paralysis of the pharynx, larynx and palate, leading to dysphagia and dysphonia; on the opposite side – pain and temperature hemihypoesthesia. Variants of this syndrome are often observed, which are manifested mainly by dizziness, nystagmus, and cerebellar ataxia [8, 9]. The second most common variant of ischemic stroke in the vertebrobasilar basin, manifested by non-isolated vertigo, is due to blockage of the anterior inferior cerebellar artery. In this case, in addition to dizziness, the following disorders are usually observed: ipsilateral paresis of the facial muscles and hearing loss, paresis of the gaze towards the focus; contralateral reduction of pain and temperature sensitivity. In addition, nystagmus, tinnitus, cerebellar ataxia, and Horner's syndrome are characteristic. Occlusion of the initial part of the artery may be accompanied by involvement of the corticospinal pathway and, consequently, hemiparesis. Isolated vertigo as a symptom of stroke is much less common. In a recent large population-based study, it was shown that stroke is caused by only 0.7% of cases of isolated vestibular vertigo. However, according to other data, 62% of patients with stroke had at least one episode of vestibular vertigo in the vertebrobasilar system, and in 19% of the examined, vertigo was the first symptom of stroke, which was later joined by other manifestations of cerebral stem or cerebellar ischemia. Another recent study showed that patients hospitalized for acute vertigo had a 2-fold higher risk of all acute cardiovascular events over the next 3 years than in the general population. It has been noted that patients hospitalized with acute vertigo will have a 3-fold higher risk of stroke in the next 4 years than in the general population [6,7]. Therefore, the relationship between cerebral vascular pathology and vertigo is not yet well understood and needs further research, and isolated vertigo may be a more frequent manifestation of cerebral ischemia, especially in groups at high risk of cerebrovascular disease. As an anatomical substrate of isolated vertigo in cerebrovascular diseases, a selective lesion of the cerebellar nodule (this area is supplied with blood by the medial branch of the posterior inferior cerebellar artery), a cerebellar fragment, a section of the brainstem in the area of the entrance of the vestibular nerve root and vestibular nuclei is distinguished. There are few descriptions of isolated vestibular vertigo, which occurs when brain structures outside the brain stem and cerebellum are affected. For example, acute isolated vertigo has been described in infarcts in the insula and left parietal lobe with spread to the supramarginal gyrus [13]. Moreover, in these cases, dizziness could be accompanied by horizontal nystagmus.

In cases where stroke leads to the formation of persistent vestibular dysfunction manifested by nystagmus, symptomatic drugs are used to reduce oscillopsia and instability [7]. So, in case of downward nystagmus caused by ischemia of the lower parts of the brainstem or bilateral damage to the cerebellar patch zone, baclofen at a dose of 5 mg 3 times a day or clonazepam at a dose of 0.5

mg 3 times a day can be effective. In stroke with damage to the medial parts of the medulla oblongata or pontomesencephalic area, upward nystagmus occurs. Such nystagmus is rarely persistent, and therefore treatment, as a rule, is not required. If it does lead to long-term oscillopsia, baclofen is prescribed at a dose of 5-10 mg 3 times a day. A stroke with damage to the area of the pons and medulla oblongata can lead to the formation of acquired pendulum-like nystagmus. In such cases, memantine 10 mg 4 times a day or gabapentin 300 mg 4 times a day are used [10]. The expediency of the widespread use of vasoactive and nootropic drugs in the treatment of patients with dizziness due to stroke is doubtful. Nevertheless, some drugs, according to the results of controlled trials, can reduce the severity of instability in this category of patients. One of these drugs is nicergoline (Sermion). The therapeutic efficacy of the drug is due to its alpha-adrenergic blocking effect and direct effect on neurotransmitter systems – noradrenergic, dopaminergic and acetylcholinergic. According to the results of a randomized, double-blind, placebo-controlled trial [8,9], the use of nicergoline at a dose of 60 mg/day for 3 months in patients with instability caused by damage to the central parts of the vestibular analyzer led to a significant improvement in the condition according to the results of the Dizziness Handicap Inventory (DGI) and the Dizziness Assessment Rating Scale (DARS). Vestibular rehabilitation is an important component of the treatment of a patient with dizziness caused by stroke [8]. Vestibular rehabilitation in stroke patients is less effective than in peripheral vestibular disorders. This is due to the fact that for full vestibular adaptation, it is necessary to preserve the cerebellum and its connections with the vestibular system, since it is it that provides a modulating effect on the vestibulo-ocular reflex and contributes to vestibular compensation. Meanwhile, it is the cerebellum that is often damaged during a stroke accompanied by dizziness. Despite this, vestibular rehabilitation in stroke patients reduces the risk of falls and improves quality of life [11]. Thus, the examination of a patient with acute first-onset vertigo should always be aimed at ruling out stroke as a possible cause of vestibular dysfunction. Dizziness in stroke is usually accompanied by other focal neurological symptoms, but in some cases it can be isolated. Differential diagnosis using anamnesis, clinical neurovestibular and neurological examination, MRI of the brain in most cases is effective and makes it possible to distinguish dizziness caused by stroke from vertigo in peripheral vestibular disorders in a timely manner.

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