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On the Differentiation of Central and Peripheral Vestibulopathies: A Literature Review

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Abstract Diagnosis of the genesis of vertigo is currently one of the most difficult tasks of medicine due to the large number of pathologies with this syndrome that are diverse in genesis. The article presents a review of the literature of recent years on the prevalence, etiopathogenesis, diagnosis, differential diagnosis, treatment and rehabilitation of vestibulopathies of central and peripheral genesis.

Keywords Central vestibulopathies, Peripheral vestibulopathies, Diagnosis, Differentiation, Treatment, Rehabilitation

1. Introduction

Dizziness is one of the leading causes of visits to neurologists and related specialists [24]. Attacks of dizziness occur at any age, and "over the course of their lives, 17–30% of people experience symptoms of non-systemic (non-vestibular) dizziness, and 3–10% experience systemic (vestibular, rotational) dizziness, with 4% consulting ENT doctors and 10% consulting neurologists" [14].

Dizziness occurs due to pathologies of the vestibular apparatus, diseases of the central and peripheral nervous system, and somatic diseases [1,23].

The prevalence of vestibular disorders in adults is 28-81 cases per 100,000 population and is characterized by a steady increase; they are the cause of 4-7% of complaints of dizziness [4].

20-30% of the adult population have experienced instability once or more in their lives, and about 4.9% of the world's population complains of instability annually.

Dizziness is also one of the most common complaints among neurologists, increasing in frequency in middle and old age [15,25].

Dizziness accounts for 5–20% of visits to a neurologist and is considered one of the leading causes of urgent hospitalization [12,16].

The most common diagnoses for dizziness in middle age are vertebral artery syndrome, and in old age – cerebrovascular insufficiency [8].

The etiological leaders of dizziness are considered to be pathologies of the peripheral vestibular analyzer, and among neurological pathologies - vestibular migraine; unfortunately, dizziness of psychogenic genesis is rarely diagnosed [6].

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The increase in the incidence of statodynamic disorders, in addition to age, is also associated with gender: women suffer 2.7 times more often than men [2,37]. In Russia, dizziness and instability have been reported in over 7 million people [12].

Thus, the sufficient prevalence of a wide group of diseases, united under the general name of "vestibulopathies", and the constant increase in their number, which is most likely due to the increase in the average life expectancy of the population around the world, makes their study a highly pressing medical and social problem.

Vestibulopathies are "a large group of pathological syndromes and diseases that develop with various pathogenetic variants of damage to the peripheral or central part of the vestibular analyzer, which performs 3 main functions - orientation in space, maintaining balance, stabilizing the image" [1,3].

Most of the genesis of dizziness and instability can be determined on an outpatient basis without additional vestibular examination, for example, BPPV – "benign paroxysmal positional vertigo" [22].

There are 4 types of dizziness: "systemic, disturbance of balance and gait (postural instability), feeling of nausea and impending loss of consciousness, and psychogenic dizziness" [39].

In 2009, the Robert Barany International Society of Otoneurology presented the characteristics of the main terms: " Vertigo (systemic, rotational dizziness) - a sensation of imaginary movement of one's own body or surrounding objects in space; Dizziness (non-systemic, non-rotational dizziness) - a sensation of disturbed spatial orientation without a false or distorted sense of movement; Imbalance or "unsteadiness (unsteadiness) is a feeling of instability of one's own body while sitting, standing or walking without leaning the body in a certain direction" [28].

Vertigo in peripheral vestibulopathies is caused by “pathologies of the peripheral part of the vestibular system with a subjective perception of rotation of surrounding objects or one’s own body, often with nausea, vomiting, sweating, hearing loss, nystagmus and balance disturbances” [3,10,17,43].

Non-systemic dizziness is often caused by central or somatic factors (vascular and psychogenic genesis, migraine), which is characterized by a subjective feeling of instability, a "floating" state, unsteadiness, "unclearness" in the perception of space, derealization of sensations, diplopia, dysarthria, weakness, headache [1,19]. Detailing the symptoms of non-systemic dizziness is of great importance for establishing a diagnosis.

The widespread prevalence of unsteadiness and dizziness is due to a wide range of different pathologies, which creates difficulties in differential diagnosis of their etiological factor [2]. The probability of incorrect diagnosis verification in patients with unsteadiness and dizziness ranges from 20-40% [12], which clearly reduces the effectiveness of their treatment and rehabilitation [22].

Balance disorders significantly reduce the quality of life (QoL) of patients by dramatically reducing their daily activities, thereby causing social maladjustment and loss of ability to work. Acute attacks of dizziness and unsteadiness are one of the top 10 reasons for urgent visits to a neurologist [35]. Most often, the genesis is benign peripheral vestibular disorders, but 3-5% of patients are diagnosed with life-threatening pathologies – stroke.

An attack of intense vertigo indicates its systemic nature and there is a high probability of a vestibular system pathology, while the sensation of the floor swaying like the rocking of a ship while walking is more characteristic of non-systemic dizziness – dizziness – transient non-rotational dizziness [12,22,29,30].

It is necessary to assess the duration of the attack: acute, episodic or chronic vestibular syndrome [29,30].

Acute vestibular syndrome (AWS) is a “clinical syndrome characterized by a single, acutely occurring episode of dizziness and/or instability, lasting from several hours to several weeks, caused by dysfunction of the vestibular-cochlear apparatus or the central nervous system” [12]. It is often a single episode, less often the first episode of pathology progression [22]. The main genesis of AWS are “vestibular neuronitis, acute labyrinthitis, traumatic vestibulopathy, stroke with damage to the central and peripheral vestibular structures” [12].

Episodic vestibular syndrome (EVS) is a "clinical syndrome characterized by transient, recurrent episodes of dizziness and/or unsteadiness lasting from a few seconds to several hours, or less commonly, days, caused by temporary, recurring dysfunction of the vestibular apparatus or central nervous system" [12]. It occurs in such pathologies as BPPV, Meniere's disease (MD), vestibular migraine, panic attacks, and transient ischemic attacks (TIA) [12].

Chronic vestibular syndrome (CVS) is “a clinical syndrome characterized by dizziness and/or instability, lasting from several months to several years, caused by a persistent, often progressive disorder of the vestibular system or central nervous system, but may also be a consequence of incomplete recovery of vestibular function after RVO or between exacerbations of CVS” [12].

Differential diagnosis of dizziness types is based on the factors of onset, duration, and frequency of attacks. In a conversation with the patient, the physician should identify factors that serve as triggers for the onset of dizziness, for example, a certain position: "lying down, turning in bed, bending over, throwing the head back, sitting up in bed, indicates positional vestibular syndrome, which is most characteristic of BPPV" [12,13,22,40].

EPS can be triggered by coughing, straining, and loud sounds; in these cases, a perilymphatic labyrinthine fistula may be the cause. Psychogenic vertigo is typically triggered by specific environments, such as a crowded street or subway. Vertigo with the onset or worsening of tinnitus and/or hearing loss in the affected ear often indicates MD [12].

The most common genesis of complaints of VN is rightfully considered to be BPPV with an incidence in 18.3% of patients, in second place is phobic postural dizziness - in 15.9% of patients, in third place is “vertigo due to hemodynamic and inflammatory pathologies of the brainstem or cerebellum” - 13% of patients [22,32].

BPPV is diagnosed by “the occurrence of an episode of vertigo with peripheral positional nystagmus in the patient during provocative tests” [11].

Giving way to BPPV as the leading pathology among the EWS, BM ranks second [6]. The diagnosis of BM is based on the sudden onset of vertigo attacks lasting up to several hours with nausea and vomiting, developing at any time of the day and regardless of the patient's position [4]. VN is often combined with increasing intensity of tinnitus, congestion and pressure in the ear; hearing decreases during an attack of BM [7]. In most patients, BM begins on one side, progressing to involve the second labyrinth in half of the patients [4,5,7]. Absolute verification of BM is possible with a dehydration test - a glycerol test. 50-70% of patients report spontaneous remissions in the first 2-8 years of the pathology [27,34].

Vestibular Neuronitis (VNT) is the third most common etiology of peripheral VN. VNT is characterized by "a severe attack of vertigo and instability, sharply increasing with movement with spontaneous horizontal-rotatory nystagmus and the illusion of movement of objects (oscillopsia), nausea and vomiting lasting from hours to several days, after the end of the attack of vertigo, instability remains lasting from weeks to several months" [9,18,21,33,40].

Neurologists often diagnose vertebrobasilar insufficiency (VBI), although most of these patients have neurological disorders that are not consistent with VBI [12,22]. VBI is a "reversible impairment of brain function caused by decreased blood supply to the area supplied by the vertebral and basilar arteries, the cause being cerebrovascular pathology, manifested by TIA or small ischemic strokes in the VBI, although a gradual increase in neurological deficit is possible" [22].

In addition, with cerebral atherosclerosis and hypertensive angiopathy of the cerebral vessels, complaints of dizziness are common, but they are more limited to instability and unsteadiness of gait, especially when turning and bending [22,31].

Pharmacotherapy of dizziness most often results in the use of various combinations of vasoactive and nootropic drugs, since the genesis of dizziness is often mistakenly associated with hemodynamic disturbances, although in the new millennium it is worth noting significant progress in the treatment of dizziness [12,20,22].

Therapy for VN includes symptomatic reduction of the intensity of dizziness, oscillopsia and other autonomic disorders, pathogenetic and/or etiotropic treatment and vestibular rehabilitation (VR) [30].

VR reduces the intensity of subjective dizziness and the risk of falls, increases balance and visual acuity when turning the head in vestibular hypofunction.

VR is effective in unilateral and bilateral vestibular hypofunction - it reduces subjective symptoms, stabilizes gaze, balance, and functional activity of patients [26], and also improves visual-spatial memory; the inclusion of habituation exercises in VR reduces negative responses in patients [36].

The effectiveness of VR depends on the age and psychological state of the patient, the timing of its onset and the experience of the physician-instructor in VG, as anxiety and depression have been proven to prolong the recovery of patients [38]. VR is also influenced by subjective "motivation, attention, interest and subjective speed of functional recovery". It is recommended to perform VG immediately after the relief of an attack of dizziness [36]. Early initiation of VG corresponds to rapid functional recovery [29].

VR is carried out in the form of classes and VG using various exercise machines and a stabiloplatform, while necessarily receiving biofeedback for timely adequate correction and consolidation of the most effective static and dynamic exercises, increasing the effectiveness of VG in the VR complex [42].

The VR stage is completed as the VR goals are achieved, stability is achieved, and a plateau is reached in the restoration of vestibular functionality for a particular patient, after which the regulations and set of VG exercises should be changed [38].

2. Conclusions

Thus, diagnosing the genesis of dizziness remains one of the most challenging tasks in medicine today due to the large number of pathologies with diverse origins associated with this syndrome.

Diagnostic difficulties are further compounded by the subjectivity of the dizziness symptom, which relies on the sensations and descriptions of the patients themselves. This often leads to overdiagnosis of hemodynamic brain pathologies as the etiological factors of dizziness, which consequently leads to low treatment effectiveness. Verifying the etiological factor of dizziness requires a thorough medical history, a clear determination of the type of dizziness, its frequency and duration, attack triggers, and associated symptoms. A neurovestibular examination should then be performed.

Most studies of dizziness have examined acute attacks of dizziness in the context of emergency care, and all have found that clinical examination is more effective than instrumental methods of investigation.

Vestibular gymnastics is an effective method for restoring vestibular function in peripheral and central vestibulopathy. It is recommended at any age for the prevention of limitations in social and physical activity, especially as an adjunct to symptomatic and pathogenetic pharmacotherapy in the VR complex, accelerating and improving the restoration of vestibular function.

The effectiveness of VR in MD and psychogenic dizziness, as well as in multisensory impairment, is controversial, but VR is an effective method for restoring vestibular functionality in the form of reducing the intensity of dizziness and increasing stability in vestibulopathies and postural phobic instability.

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