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VESTIBULAR DISORDERS IN DIABETES MELLITUS (LITERATURE REVIEW)

G.U.Lutfullaeva M.T.Nasretdinova

Samarkand State Medical University

Annotation: Chronic hyperglycemia in diabetes is accompanied by damage, dysfunction and insufficiency of various organs, especially the eyes, kidneys, nerves, heart and blood vessels. predominant damage to the auditory system in DM, explaining this by the lower resistance of the organ of Corti to hypoxia.

Key words: diabetes mellitus, vestibular disorders, demyelination

Relevance of the problem: Diabetes mellitus (DM) is a disease that remains of significant clinical relevance. The number of people with diabetes in the world is growing rapidly, which represents a serious global health problem affecting the population of both developed and developing countries. This is due to changes in the lifestyle and diet of modern people, high levels of stress and an increase in the percentage of obese people. It is no coincidence that diabetes is called a disease of civilization [1].

Diabetes mellitus (DM) is a group of metabolic diseases characterized by chronic hyperglycemia, which results from impaired insulin secretion, insulin action, or both. Chronic hyperglycemia in diabetes is accompanied by damage, dysfunction and failure of various organs, especially the eyes, kidneys, nerves, heart and blood vessels. Type 1 diabetes is divided into 2 forms: immune-mediated and idiopathic, in which destruction of pancreatic β-cells occurs, usually leading to absolute insulin deficiency [2].

In conditions of excess glucose in the blood, it becomes necessary to connect alternative pathways for its utilization. Activation of the polyol pathway leads to the accumulation of sorbitol and fructose in peripheral nerve cells, which increases cellular osmolarity and fluid entry into the nerve. [1]. In addition, NADPH depletion, the development of oxidative stress and a decrease in the synthesis of nitric oxide (NO) - a powerful local vasodilator - occur. Excessive glycolysis can overload the mitochondrial electron transport chain and produce reactive oxygen species. Glucose utilization through the hexosamine pathway is associated with inflammatory cell damage [8,9]. Another consequence of hyperglycemia is the formation and accumulation of advanced glycation end products. Non-enzymatic glycation of serum proteins, collagen, and nerve fibers disrupts their biological function. Dyslipidemia, in turn, can lead to weakened endoneurial blood flow, which also contributes to the pathogenesis of DPN. Thus, a cascade of pathological reactions is formed that contribute to the development of oxidative stress, demyelination of nerve fibers, axonopathy and the launch of apoptosis of nerve cells.

In older people, diabetes is an independent risk factor for falls [4,5]. DPN makes a significant contribution to the development of instability [5,9]. However, most likely this is not the

only cause of balance disorders in diabetes [13]. Postural stability is the ability to maintain balance when changing posture. It largely depends on information entering the central nervous system from the vestibular, visual and proprioceptive systems. Moreover, among these three afferent systems, it seems that the vestibular one plays the main role in maintaining postural stability, especially in difficult conditions - with insufficient lighting or walking on an uneven surface [2, 8]. The importance of vestibular dysfunction in the development of postural instability in diabetes is largely underestimated. Meanwhile, a large epidemiological study showed that vestibular disorders in patients with diabetes are almost twice as common as in the general population [10]. Studying the role of vestibular disorders in the development of postural instability in diabetes is largely difficult due to the complexity and laboriousness of objectifying vestibulopathies. It is even more difficult to assess the contribution that vestibular dysfunction makes to postural stability disorders in diabetes [5,6,8]. Despite the high incidence of vestibular disorders in diabetes, classic vestibular vertigo is no more common than in the general population, which may be due to symmetrical damage to the vestibular system. Clarifying the role of vestibular disorders in the development of instability in patients with diabetes has not only important theoretical, but also practical significance, since it can help optimize the treatment of balance disorders and reduce the risk of falls in this group of patients. The vestibular system is represented by five final sense organs located in the inner ear on each side. Ampullary receptors are located in the expanded part of the anterior, posterior and horizontal semicircular canals - the ampulla. Otolith receptors are located in the vestibule of the labyrinth and are represented by elliptical and spherical sacs. The ampullary receptors of the semicircular canals perceive angular accelerations, and the otolith receptors perceive linear ones. The afferent pathway of these end organs - the superior and inferior branches of the vestibular part of the vestibular-cochlear nerve - ends on the vestibular nuclei of the brain stem. In turn, the vestibular nuclei are closely interconnected with various parts of the brain and spinal cord through vestibulo-oculomotor, vestibulocerebellar, vestibulothalamic and vestibulospinal collaterals. As is known, glucose metabolism significantly affects the physiology of the inner ear, where metabolism is extremely active [1, 3, 6]. Thus, S.F. Myers and co-authors in a number of studies assessed morphological changes in the inner ear in rats with experimental diabetes. According to the data obtained, metabolic stress caused by hyperglycemia leads to selective degeneration of type 1 hair cells in the sac [6], demyelination of the vestibulocochlear nerve [7], accumulation of extracellular matrix and an increase in the number of lysosomes and lipid droplets in the connective tissue of the elliptic and spherical sacs [6]. The predominant damage to type 1 hair cells in diabetes is explained by their higher sensitivity to damage in general and to hypoxia in particular. As a possible explanation for the selective damage to hair cells, the study examined the hypothesis that different cells absorb and process glucose differently. In this case, cells that are unable to sufficiently limit the influx of glucose under conditions of hyperglycemia become especially vulnerable to the damaging effects of glucose. Not only metabolic disorders, but also hypoxia is an important mechanism of damage to the vestibular system in diabetes.

To date, a significant amount of data has accumulated indicating an association between diabetes mellitus and sensorineural hearing loss (SNHL). However, the nature of the pathogenetic relationship between these diseases remains unclear. The authors found predominant damage to the auditory system in diabetes, explaining this by the lower resistance of the organ of Corti to hypoxia [4,7]. With the introduction into practice of instrumental methods for studying the vestibular system, it became possible to objectify previously obtained information about damage to the inner ear in patients with diabetes. Thus, video and electronystagmography using video recording tracks eye movements and allows one to study nystagmus (spontaneous, latent,

positional) and central mechanisms of gaze control (smooth visual pursuit, randomized saccades, optokinetic nystagmus). The video impulse test, based on measuring the speed of head rotation and simultaneous eye movement in the opposite direction during visual fixation, is intended to study the vestibulo-ocular reflex (VOR).

At the same time, signs of peripheral vestibular dysfunction were identified in 60% of patients. The results of other studies, on the contrary, indicate the central nature of vestibular dysfunction. In addition to vestibular gymnastics, the achievement of vestibular compensation can be accelerated with the help of medications. Currently, many relatively safe and effective drugs are available that can provide an auxiliary effect in the treatment of dizziness and unsteadiness. Betahistine dihydrochloride is a histamine-like drug that acts as a partial H1 receptor agonist and a more potent H3 receptor antagonist. It improves microcirculation in the inner ear [8], enhances the release of histamine in the central nervous system [11], which has a positive effect on restoring the balance of activity of the vestibular nuclei on both sides and central vestibular compensation [2,11], and has a potential inhibitory effect at the level of the final organs of the vestibular system, thereby helping to reduce symptoms of dizziness and faster recovery from vestibular loss. Ginkgo biloba has also been used for many years for vestibular disorders. Numerous experimental and clinical studies have proven its positive effect on neuronal plasticity, cognitive functions and the rate of recovery in various vestibular diseases [3,6,9]. Caffeine can also accelerate vestibular compensation, possibly by stimulating the patient's physical activity [9,13]. However, its distribution in clinical practice is limited by the high risk of side effects.

Thus, the data available today allow us to talk about quite a variety of options for damage to the vestibular system in diabetes. The mechanisms of vestibular dysfunction are mainly reduced to dysmetabolic and hypoxic processes. At the same time, the depth of damage to the vestibular system in diabetes, as well as the contribution of vestibular dysfunction to the development of postural instability, falls and a decrease in the quality of life of patients require further research.

References

1. Azimova A.A., Doskina E.V., Zhiznevsky D.V. and others. Vestibular disorders in diabetes mellitus. Effective pharmacotherapy. 2022; 18 (43): 72–77.

2. Algorithms for specialized medical care for patients with diabetes mellitus. Edited by I.I. Dedova, M.V. Shestakova, A.Yu. Mayorova.

3.. Valieva S. Sh. and others. Study of vestibular function in Meniere's disease // Issues of science and education. – 2021. – No. 14 (139). – P. 62-69.

4. Valieva S. Sh. and others. Diagnostic tactics for studying the nasal cavity in children with congenital cleft palate //European science. – 2021. – No. 3 (59). – pp. 49-52.

5. Zamergrad M.V. Rehabilitation for diseases of the vestibular system. Neurology, neuropsychiatry, psychosomatics. 2013; 1:18–22.

6. Nasretdinova M.T., Karabaev Kh.E. Improving diagnostic methods in patients with dizziness, Otorhinolaryngology. Eastern Europe 7 (2), 194-198.

7. Nasretdinova M.T., Karabaev Kh.E. Dizziness in ENT practice innovative technologies in pediatric medicine of the North Caucasus Federal District 216-219.

8. Nasretdinova M.T. Changes in stabilometric parameters in patients with systemic dizziness Otorhinolaryngology. Eastern Europe 9 (2), 135-139.

9. Nasretdinova M. et al. Diagnostic value of the glyceryl test for Meniere's disease // Journal of Dentistry and Craniofacial Research. – 2021. – T. 2. – No. 1. – pp. 34-37.

10. Nasretdinova M., Narzullaev I., Normuradov N. Circulatory insufficiency in the

vertebrobasilar system and peripheral cochleovestibular syndromes // Journal of Dentistry and Craniofacial Research. - 2021. - T. 2. – No. 3. - P. 54-58.

11. Omonov Sh.E., Nasretdinova M.T., Nurmukhamedov F.A. Optimization of methods for determining ear noise in various pathologies. Bulletin of the Kazakh National Medical University, 67-68.

12. Tovazhnyanskaya E.L. Diabetic peripheral polyneuropathy: modern aspects of metabolic therapy. Health of Ukraine. 2010. Thematic issue, October.

13. Fabrichnova A.A., Kulikov D.A., Misnikova I.V. and others. Changes in the rheological properties of blood in diabetes mellitus. Endocrinology. News. Opinions. Education. 2018; 2 (23): 26–34.

14. Agrawal Y., Carey J.P., Della Santina C.C., et al. Diabetes, vestibulardysfunction, and falls: analyses from the National Health and Nutrition Examination Survey. Otol. Neurotol. 2010; 31 (9): 1445–1450.

15. Aladag I. et al., 2009; Boomsma L.J. et al., 1998; Fukushima H. et al., 2006; Kakarlapudi V. et al., 2003

16. Bamanie, A.H. et al., 2011; Mitchell, P. et al., 2009; Mozaffari, M. et al., 2010; Sunkum, A.J. et al., 2013; Uchida, Y. et al., 2010

17. Bonnet C.T., Ray C. Peripheral neuropathy may not be the only fundamental reason explaining increased sway in diabetic individuals. Clin. Biomech. (Bristol, Avon). 2011; 26 (7): 699–706.

18. Biurrun O., Ferrer J.P., Lorente J., et al. Asymptomatic electronystagmographic abnormalities in patients with type I diabetes mellitus. ORL
J. Otorhinolaryngol. Relat. Spec. 1991; 53 (6): 335–338.