Vertebral Artery Syndrome: Literature Review With A Focus On Etiology, Classification, Diagnosis, And Treatment

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ABSTRACT

Vertebral artery syndrome (VAS) is a relevant interdisciplinary problem in modern neurology caused by impaired blood flow in the vertebral arteries and their branches. The review examines current concepts of the epidemiology, risk factors, and pathogenesis of VAS, with special attention paid to its association with cervical spondylosis and lifestyle. The main classifications (etiopathogenetic, clinical, topical, by stages and mechanisms), clinical variants of the course, diagnostic criteria according to N.V. Vereshchagin, as well as the algorithm of complex examination are presented. The principles of conservative and surgical treatment, rehabilitation, and prevention are highlighted. The necessity of early diagnosis, a differential approach, and interdisciplinary interaction to improve outcomes and quality of life of patients is emphasized.

Keywords: Vertebral artery syndrome, cervical spondylosis, vertebrobasilar insufficiency, dizziness, cervicogenic headache, diagnosis, classification, treatment

Relevance

Vertebral artery syndrome (VAS) remains one of the most common and clinically significant forms of vertebrobasilar insufficiency [9, 52]. In recent decades, against the background of population aging, widespread physical inactivity, prolonged work with gadgets, and occupational loads, the frequency of cervical spondylosis and associated VAS has increased significantly [2, 10, 13, 23]. The pathology is acquiring the features of a "silent epidemic," significantly reducing patients' quality of life, limiting work capacity, and increasing the risk of developing transient ischemic attacks and ischemic stroke in the vertebrobasilar basin [11, 45, 52].

Despite its significant prevalence, VAS often remains underdiagnosed due to the nonspecific nature of the clinical picture and the absence of generally accepted international diagnostic criteria [12, 34]. In domestic neurology, the term VAS retains wide practical use as a convenient clinical syndrome [4, 14, 31]. The relevance of the problem is due to the need to systematize modern data on the etiology, pathogenesis, classification, diagnosis, and treatment of VAS, taking into account the characteristics of the disease course in various categories of patients, including military personnel and working-age individuals [3, 13, 23].

The aim of this review is to analyze the current state of the problem of vertebral artery

syndrome based on data from domestic and foreign literature, to systematize information on etiology, risk factors, pathogenesis, classifications, clinical variants, principles of diagnosis, treatment, and prevention of VAS [1, 15, 34].

Materials and Methods The review was performed based on the analysis of scientific literature published between 2018 and 2025. The search for sources was conducted in the databases PubMed, MEDLINE, eLibrary.ru, CyberLeninka, as well as in leading Russian and international neurology journals [15, 34]. The following keywords were used: "vertebral artery syndrome", "vertebrobasilar insufficiency", "cervical spondylosis", "cervicogenic dizziness", "vertebrogenic compression" [16, 60].

The analysis included original articles, reviews, clinical guidelines, and monographs devoted to the epidemiology, pathogenesis, diagnosis, and treatment of VAS. Special attention was paid to the works of N.V. Vereshchagin and co-authors, as well as modern international studies [4, 17, 34, 52]. A total of more than 120 sources were analyzed, of which 64 of the most relevant were selected. Priority was given to publications with a high level of evidence [18, 57]. The data were summarized and systematized in accordance with modern requirements of evidence-based medicine [19, 53].

Results Vertebral artery syndrome (VAS) is a complex of clinical manifestations caused by impaired blood flow in the vertebral artery. The most common cause is compression or irritation of the vessel at the level of the cervical spine [20, 29, 10]. In recent years, against the background of rapid technological development and changes in lifestyle, this pathology has acquired the features of a silent epidemic, which requires increased attention from the medical community [2, 21, 23].

Epidemiology and Global Burden Modern studies show that cervical spondylosis, which is one of the leading causes of VAS development, is becoming an increasingly common disease, especially among aging populations [2, 22, 23]. The demographic shift in many countries around the world leads to an increase in the number of people at risk of degenerative changes in the cervical spine [23, 35]. Particular concern is raised by the increasing prevalence of cervical spondylosis in developed countries, where prolonged static postures, computer work, and the use of mobile devices significantly increase the load on the cervical spine [2, 24, 33].

Professional risk factors also play an important role in the development and progression of cervical spondylosis and, consequently, vertebral artery syndrome [3, 13, 23]. VAS is increasingly viewed as a disease of civilization, reflecting a close connection with the modern lifestyle [2, 25, 40]. Numerous works emphasize that cervical spondylosis is turning into a growing public health problem [2, 26].

Risk Factors and Etiology Population aging and increased life expectancy are the leading factors in the growing prevalence of cervical spondylosis and vertebral artery syndrome (VAS) [2, 29, 35]. Every year, more and more people face this problem, which creates a significant burden on healthcare systems [23, 30].

In addition to age-related changes, professional factors play an important role. Prolonged stay in uncomfortable positions, monotonous neck movements, and work in conditions of vibration significantly increase the risk of developing cervical spondylosis [3, 13, 23, 31]. The risk group includes office workers, drivers, surgeons, and other specialists experiencing increased load on the cervical spine [31, 40]. These factors, combined with degenerative changes, often lead to compression of the vertebral artery [10, 20, 29].

Modern technologies also have a direct negative impact on neck health. Prolonged use of gadgets, computer work, and poor posture contribute to the acceleration of degenerative processes [2, 24, 33]. Many authors characterize cervical spondylosis as a modern pandemic of a sedentary lifestyle [2, 25].

Assessment of the true prevalence of VAS is difficult due to the lack of unified diagnostic criteria, the nonspecific nature of symptoms, and frequent concomitant pathology [12, 30, 34]. Nevertheless, available data indicate that VAS is more common in people over 40 years of age, although in recent years there has been a

trend toward rejuvenation of the disease [2, 35]. Some studies note a slightly higher prevalence among women in the postmenopausal period [36].

Risk factors for VAS are usually divided into several groups. Degenerative-dystrophic factors include cervical osteochondrosis, spondyloarthrosis, and spinal instability [10, 37]. Vascular factors include atherosclerosis, arterial hyper- and hypotension, vasculitis, and congenital anomalies of the vertebral arteries [38, 61]. Traumatic factors include direct neck injuries and iatrogenic damage [39]. Other significant factors include a sedentary lifestyle, chronic stress, smoking, obesity, and certain types of professional activities [13, 23, 40, 43].

Thus, vertebral artery syndrome is a multifactorial condition, the basis of which is most often degenerative changes in the cervical spine and vascular disorders [10, 41, 45]. A deep understanding of risk factors and etiology allows timely identification of at-risk patients, effective prevention, and treatment [1, 42]. Special attention should be paid to individuals with concomitant diseases and those leading a sedentary lifestyle [2, 43]. Further research and standardization of diagnostic criteria will contribute to improved management of this common syndrome [34, 44, 57].

Pathogenesis of VAS

The central place in the etiology and pathogenesis of vertebral artery syndrome (VAS) is occupied by cervical spondylosis - a degenerative-dystrophic disease of the cervical spine [45, 10, 20]. Manifesting as osteophytes, protrusions of intervertebral discs, and ligamentous hypertrophy, spondylosis becomes the main cause of vertebral artery compression [20, 29, 46]. These age-related structural changes lead to narrowing of the vessel lumen, decreased elasticity, and the development of cerebral ischemia [38, 47].

The main pathogenetic mechanism of VAS is compression or irritation of the vertebral artery caused by bone growths (osteophytes), disc herniations, spondylolisthesis, or instability of the cervical spine [24, 48]. Aging is one of the key factors accelerating these processes [2, 29, 49].

Despite its growing prevalence, vertebral artery syndrome often remains underdiagnosed. This is primarily due to the nonspecific nature of symptoms that can mimic other neurological and vascular diseases [12, 50, 53]. In this regard, the standardization of diagnostic criteria is acquiring the character of a global imperative [34, 51, 57]. The problem of underestimation and diagnostic difficulties of VAS leads to an increase in the disease burden [45, 52]. Many authors emphasize the need to increase awareness among physicians and patients about the characteristic manifestations (headache, dizziness, tinnitus, visual and coordination disorders), which will help reduce delays in providing care [7, 9, 53].

International cooperation of scientists, clinicians, and healthcare organizations plays a decisive role in improving approaches to the prevention, diagnosis, and treatment of VAS [54, 62]. The development of unified criteria, the introduction of modern visualization methods (MRI, CT, Doppler ultrasound), and a comprehensive assessment of patients are key directions for increasing the effectiveness of management of this syndrome [12, 34, 55, 60].

Classification of Vertebral Artery Syndrome

Vertebral artery syndrome (VAS) is a complex of symptoms that arise due to impaired blood flow in the vertebral arteries and their branches, which supply blood to the brainstem, cerebellum, occipital lobes, inner ear, and upper parts of the spinal cord [12, 56, 60]. The classification of VAS is of great importance for understanding the etiology, pathogenetic mechanisms, clinical picture, diagnosis, and selection of optimal treatment tactics [15, 21, 57].

I. Main etiopathogenetic classification identifies the following forms.

Vertebrogenic VAS is the most common variant (up to 90% of cases) and is caused by pathology of the cervical spine leading to compression or irritation of the vertebral artery and the surrounding sympathetic plexus [10, 20, 29, 59]. It includes degenerative-dystrophic changes (osteochondrosis with disc herniations and osteophytes, spondylosis, spondyloarthrosis), congenital anomalies (Kimmerle anomaly, vertebral artery

hypoplasia, platybasia, basilar impression), post-traumatic conditions, muscular-tonic syndromes, and inflammatory diseases (rheumatoid arthritis, ankylosing spondylitis) [10, 23, 37, 60].

Non-vertebrogenic VAS is associated with causes independent of the cervical spine. This group includes atherosclerosis and thrombosis of the vertebral artery, embolism, vasculitis, as well as extravascular compression by tumors, lymph nodes, or scar changes [61, 38]. A separate place is occupied by hemodynamic disorders (a sharp drop in systemic pressure against the background of existing arterial pathology) and mixed VAS, in which vertebrogenic and non-vertebrogenic factors are combined. The latter variant occurs especially often and is characterized by the most severe course [41, 62].

II. Classification by Clinical Syndromes This classification reflects the main clinical variants observed in VAS. One patient often has a combination of several syndromes [15, 63].

1. **Cervicocranialgic (cervicogenic) syndrome** is manifested by headache in the occipital region and neck with spread to the forehead, temples, and crown. The pain intensifies with head movements, static tension, palpation of muscles and spinous processes. It is often accompanied by non-systemic dizziness (lightheadedness, unsteadiness) and includes Barre-Lieu syndrome (posterior cervical sympathetic syndrome) [8, 9, 64].
2. **Vestibular (cochleovestibular) syndrome** is characterized by systemic dizziness (vertigo), noise or ringing in the ears (tinnitus), hearing loss (mainly sensorineural hearing loss at low frequencies), unsteadiness, nausea, and vomiting. Symptoms are usually provoked by head turns [36, 59].
3. **Vestibulo-atactic syndrome** includes pronounced unsteadiness and staggering when walking, coordination disorders (intention tremor, missing), and nystagmus. Dizziness may be less pronounced than ataxia; it is often caused by cerebellar ischemia [56, 63].
4. **Ophthalmic syndrome** is manifested by rapid eye fatigue, a sensation of "sand," conjunctival redness, pain in the eye sockets, as well as transient visual disturbances (sparks, flashes, fog, flickering, photopsias, less often - diplopia or decreased visual acuity) [34, 35].
5. **Syndrome of autonomic dystonias** includes hot or cold flushes, chills, cold extremities, lability of blood pressure, sweating, a feeling of shortness of breath, cardialgias, sleep disturbances, irritability, and anxiety [7, 45].
6. **Syncopal syndrome** is characterized by short-term loss of consciousness provoked by sharp turns or tilting of the head (including Unterharnscheidt syndrome). Weakness and dizziness are noted after the episode [52].
7. **Atactic syndrome (drop attacks)** is manifested by sudden falls without loss of consciousness, provoked by tilting the head back, with preserved consciousness ("legs give way") [52].
8. **Transient ischemic attacks (TIAs)** in the vertebrobasilar basin are acute focal neurological symptoms (weakness of the limbs, speech, swallowing, vision, coordination disorders, pronounced dizziness) that completely regress within 24 hours. This condition carries a high risk of stroke [6, 52].
9. **Brainstem syndrome** reflects ischemia of the medulla oblongata and includes a combination of cranial nerve lesions (dysarthria, dysphagia, nystagmus, ptosis), pyramidal signs, sensory and autonomic disorders, as well as ataxia [52].

Such a clinical classification helps in the accurate assessment of symptoms and the choice of individual treatment tactics [14, 21].

III. Classification by Topical Principle (Level of Lesion) This classification reflects the

anatomical localization of the pathological process [34].

The **extracranial level** (outside the cranial cavity) includes lesions in the canal of the transverse processes of the cervical vertebrae (C2–C6) - the most common localization in vertebrogenic VAS (osteophytes, disc herniations, muscle spasm), as well as the level of the atlas (C1) and the foramen magnum (Kimmerle anomaly, C1–C2 instability, basilar impression). In addition, lesions at the mouth of the vertebral artery (atherosclerosis, hypoplasia) and along the course of the artery in the soft tissues of the neck (tumors, lymph nodes, scars, scalene muscle spasm) are distinguished [12, 29, 35].

The **intracranial level** (inside the cranial cavity) covers lesions of the intracranial portion of the vertebral artery (atherosclerosis, aneurysms, dysplasias, vasculitis), the basilar artery (atherosclerosis, thrombosis, embolism), and the cerebellar arteries (posterior inferior, anterior inferior, superior cerebellar arteries) [12, 38].

IV. Classification by Stages and Forms of Course According to stages (according to N.V. Vereshchagin and co-authors), the following are distinguished: **Stage I (dystonic)** - functional disorders of the tone of the vertebral artery and sympathetic plexus. It manifests as headache, dizziness, and autonomic symptoms during provocations. Changes in blood flow according to ultrasound Doppler are minimal and are detected mainly during functional tests [17].

Stage II (ischemic) is characterized by organic changes in the arterial wall (stenosis, deformation) and persistent regulatory disorders. Symptoms are more pronounced and constant, including transient ischemic attacks. Instrumentally, stenosis >30%, asymmetry, and a decrease in linear blood flow velocity are detected [18].

Stage III (cerebrovascular accident) includes transient disorders (TIAs), minor stroke (regression within 3 weeks), and completed ischemic stroke in the vertebrobasilar basin [19].

According to the forms of course, remitting (recurrent), progressive, and stable (stationary) forms are distinguished, as well as acute development in the form of TIA or stroke [20].

This multilevel classification facilitates understanding of the pathogenesis and clinical picture and allows individualization of diagnosis and treatment of patients with VAS [14, 21].

V. Classification by Predominant Mechanism According to the predominant pathogenetic mechanism, three main variants of VAS are distinguished [22].

The **angiospastic variant** is characterized by the predominance of reflex spasm of the vertebral artery, which most often occurs upon irritation of the sympathetic plexus against the background of muscular-tonic syndromes or Barre-Lieu syndrome [23].

The **compressive-irritative variant** is primarily associated with mechanical compression of the artery (by osteophytes, Kimmerle anomaly, tumors) in combination with simultaneous irritation of the periarterial sympathetic plexus [24].

The **occlusive-stenotic variant** is determined by organic narrowing of the vertebral artery lumen due to atherosclerosis, thrombosis, or pronounced hypoplasia [25].

Diagnostic Criteria for VAS (according to N.V. Vereshchagin and co-authors) The diagnosis of vertebral artery syndrome requires a combination of the following criteria [4, 26]:

- **Clinical signs** - the presence of characteristic symptoms (headache, dizziness, tinnitus, visual disturbances, autonomic disorders, TIAs, etc.), provoked or intensified by head turns or tilting [27];
- **Signs of cervical spine pathology** - data from X-ray, CT, or MRI confirming osteochondrosis, spondylosis, anomalies, or instability [10, 28];
- **Signs of impaired blood flow in the vertebral arteries** - data from ultrasound Doppler with functional tests (decrease in linear blood flow velocity, asymmetry, increase in resistance indices during rotation/retroflexion of the head), MR- or CT-angiography (stenosis, deformation, hypoplasia,

extravascular compression) [12, 29, 34, 55];

- **Exclusion of other diseases** - symptoms should not be explained by another pathology (tumor, multiple sclerosis, Meniere's disease, epilepsy, cardiogenic syncope, psychogenic disorders) [26, 30].

It is important to note that in the modern international ICD-11 classification, the term "vertebral artery syndrome" as an independent nosological unit is absent. Symptoms are coded according to the underlying cause (cervical osteochondrosis, cervicogenic headache, transient ischemic attack, vertebrobasilar insufficiency, etc.). Nevertheless, in domestic neurological practice, the term VAS retains wide use as a convenient clinical syndrome that unites manifestations of pathology of the vertebral arteries and the cervical spine. Understanding the multilevel classification of VAS remains extremely important for accurate diagnosis and rational treatment of patients [14, 21, 31].

Clinical Variants of the Course of Vertebral Artery Syndrome The clinical picture of vertebral artery syndrome (VAS) is characterized by significant diversity. Patients often have a combination of several syndromes [32].

Basilar migraine (migraine with brainstem aura) is characterized by intense throbbing headache, predominantly in the occiput, with possible spread to the entire head. The key feature is the aura (5–60 minutes), including visual (photopsias, hemianopsia, metamorphopsia), brainstem (systemic dizziness, ataxia, dysarthria, diplopia), and other neurological symptoms. Diagnosis is based on ICHD-3 criteria with mandatory neuroimaging to exclude organic pathology. Treatment includes prevention (beta-blockers, anticonvulsants) and attack relief [33, 39].

Barre-Lieu syndrome (posterior cervical sympathetic syndrome, "cervical migraine") is caused by irritation of the sympathetic plexus of the vertebral artery in cervical spine pathology. It manifests as dull pressing headache in the occiput, crown, and orbital area ("helmet"), intensifying with head movements, static load, and palpation. Autonomic symptoms (tinnitus, lacrimation, dry eyes) are characteristic. Diagnosis is based on the connection with cervical pathology and the positive effect of diagnostic blocks. Treatment is conservative: NSAIDs, muscle relaxants, physiotherapy, exercise therapy, and orthopedic regimen [8, 9, 64].

Vestibulo-atactic syndrome is associated with ischemia of the cerebellum and vestibular nuclei. The main manifestations are systemic dizziness, pronounced ataxia, unsteadiness, intention tremor, and nystagmus. Treatment includes etiotropic therapy, vasoactive and neurometabolic drugs, as well as vestibular rehabilitation as a key method [56, 63].

Ophthalmic syndrome is manifested by asthenopia, a sensation of "sand" in the eyes, photopsias, pain in the eye sockets, and transient visual disturbances. It develops due to ischemia of the occipital cortex or irritation of sympathetic fibers. Treatment is aimed at the underlying disease and symptomatic correction (artificial tears, visual regimen) [34, 35].

Vestibulocochlear syndrome is characterized by tinnitus, sensorineural hearing loss (mainly low-frequency), and paroxysmal dizziness. Diagnosis includes audiometry, vestibular tests, and assessment of vertebral artery hemodynamics. Beta-histine, vasoactive drugs, and vestibular rehabilitation play a leading role in therapy [36, 59].

Syndrome of autonomic disorders includes blood pressure lability, hot flushes, cardialgias, respiratory and thermoregulatory disorders, as well as asthenodepressive manifestations. Treatment requires a comprehensive approach with correction of autonomic tone and mandatory VAS therapy [7, 45].

Transient ischemic attacks (TIAs) in the vertebrobasilar basin are acute focal symptoms (dizziness, ataxia, visual and speech disturbances, sensory disorders) that completely regress within 24 hours. This condition carries a high risk of stroke and requires urgent prevention (antiplatelet agents, statins, antihypertensive therapy) [6, 52].

Unterharnscheidt syndrome is manifested by short-term syncope during head turns or tilting due to critical brainstem ischemia. Drop attacks

are characterized by sudden falls without loss of consciousness with preserved consciousness [52].

All clinical variants of VAS share common symptoms: headache, dizziness, tinnitus, and visual disturbances. Early recognition and comprehensive treatment significantly improve the prognosis and quality of life of patients [7, 45].

Key Conclusions Most clinical syndromes in VAS represent manifestations or complications of a single pathogenetic process - impaired blood supply to the posterior parts of the brain against the background of cervical spine pathology [10, 45].

Differential diagnosis is of decisive importance, especially in life-threatening conditions (TIAs, syncope, drop attacks). It is necessary to exclude stroke, tumors, epilepsy, cardiac pathology, and primary vestibular disorders [26, 30, 53].

Despite the variety of forms, VAS is characterized by common symptoms: headache (aching or throbbing, predominantly in the occiput with radiation to the temples and forehead, intensifying with head movements), dizziness of varying intensity, tinnitus, hearing impairment, as well as visual disorders ("sand in the eyes," photopsias, flashes, darkening) [7].

Without treatment, the disease progresses to the ischemic stage with the development of transient ischemic attacks, after which patients note pronounced weakness, headache, tinnitus, and visual disturbances [6, 18].

Diagnosis of VAS should be comprehensive and include neurological examination, neuroimaging (MRI/MRA), ultrasound Doppler with functional tests, vestibular tests, audiometry, and ECG/Holter monitoring [12, 29, 34, 35, 55].

Treatment is complex and individual in nature. Etiotropic therapy is aimed at eliminating the cause (exercise therapy, physiotherapy, manual therapy, and, if necessary, surgical decompression of the vertebral artery). Pathogenetic treatment includes vasoactive drugs and neuroprotection, symptomatic treatment - relief of pain, dizziness, and autonomic disorders. Vestibular rehabilitation and exercise therapy play an important role. In case of TIAs, urgent stroke prevention is necessary; in case of syncope and drop attacks - measures to prevent injuries [6, 11, 36, 50, 63].

The prognosis varies from favorable (in ophthalmic syndrome and Barre-Lieu syndrome with adequate treatment) to serious (high risk of stroke in TIAs, injuries in drop attacks, progression of hearing loss). Early seeking of medical attention and systematic therapy significantly improve outcomes and quality of life of patients [45, 63].

Diagnosis of Vertebral Artery Syndrome (VAS) Diagnosis of VAS is a complex multi-stage process that requires a comprehensive approach. The task is not only to confirm impaired blood flow in the vertebral arteries, but also to establish its cause, localization, severity, and to exclude diseases with similar symptoms. The diagnosis is made only when clinical signs, data on cervical spine pathology, and confirmed hemodynamic disturbances are combined [4, 26, 29].

Clinical diagnosis begins with a thorough history taking. Particular attention is paid to detailing complaints: the nature and localization of headache, features of dizziness (systemic or non-systemic), cochlear and visual symptoms, autonomic manifestations, coordination disorders, fainting or drop attacks, as well as provoking factors (head movements, static load, stress). The dynamics of the disease, concomitant pathology, and social and household history are taken into account [7, 27].

Neurological examination includes assessment of cranial nerves (nystagmus, diplopia, hearing and swallowing disorders), motor and sensory spheres, coordination, meningeal signs, as well as examination of the cervical spine (palpation, range of motion). Provocative tests are key, primarily the De Kleyn test (rotation and extension of the head), compression test of the vertebral artery, and hyperventilation test [27, 29].

Instrumental Research Methods Neuroimaging plays a leading role in detecting pathology of the cervical spine and brain. X-ray of the cervical spine allows assessment of the condition of bone structures (osteophytes, uncovertebral arthrosis, decreased disc height, Kimmerle anomalies, platybasia, subluxations,

and instability), especially when performing functional images in flexion and extension. Computed tomography (CT) provides more detailed visualization of bone changes and stenosis of the vertebral artery canals, while contrast-enhanced CT angiography is highly effective for detecting stenoses, thromboses, and extravascular compression [12, 34].

Magnetic resonance imaging (MRI) of the cervical spine is considered the "gold standard" for assessing soft tissues - intervertebral discs, ligaments, muscles, spinal cord, and roots. MR angiography (MRA) with or without contrast allows visualization of the course of the vertebral arteries, detection of stenoses, hypoplasia, anomalies, and dynamic compression during functional tests. MRI of the brain is mandatory to exclude stroke, tumors, multiple sclerosis, and signs of chronic ischemia in the vertebrobasilar basin [10, 12, 34, 35].

Ultrasound methods provide assessment of hemodynamics in the vertebral arteries. Ultrasound Dopplerography (USDG) of the extracranial segments determines localization, diameter, blood flow velocity, and the presence of plaques. Functional tests are key, primarily the De Kleyn test (rotation and extension of the head), in which a decrease in linear blood flow velocity by more than 50% or asymmetry over 30% confirms the hemodynamic significance of compression. Transcranial Dopplerography (TCDG) assesses intracranial blood flow and detects microembolic signals [29, 55].

Additional methods include videonystagmography (VNG) for differentiation of vestibular disorders, stabilography for balance assessment, audiometry to confirm hearing loss, as well as ECG, Holter monitoring, and echocardiography to exclude cardiac pathology. Rheoencephalography and ophthalmodynamometry currently have limited value [36, 55, 59].

Differential Diagnosis The symptoms of VAS are nonspecific and require careful exclusion of other diseases. Among peripheral vestibular disorders, Meniere's disease, vestibular neuritis, benign paroxysmal positional vertigo (BPPV), and labyrinthitis are differentiated. It is important to exclude central neurological conditions - ischemic stroke in the vertebrobasilar basin, multiple sclerosis, tumors of the posterior cranial fossa, parkinsonism, and normal pressure hydrocephalus. Among cardiovascular causes, cardiogenic syncope, orthostatic hypotension, and atherosclerosis of the carotid arteries are considered. It is also necessary to differentiate psychogenic disorders (panic attacks), basilar migraine, cervicogenic headache, and metabolic disorders [26, 30, 53, 59].

A comprehensive approach combining clinical data, neuroimaging, and functional tests allows accurate verification of the VAS diagnosis and determination of the optimal treatment tactics [4, 34, 35].

Diagnostic Criteria for VAS (according to N.V. Vereshchagin and co-authors, adapted) For a reliable diagnosis of vertebral artery syndrome, the simultaneous presence of three mandatory criteria is required:

1. **Clinical criterion** - a characteristic symptom complex (headache, dizziness, tinnitus, visual and autonomic disorders, coordination disorders, TIAs, syncope, or drop attacks), provoked or intensified by head turns and tilting [27].
2. **Vertebrogenic criterion** - data from X-ray, CT, or MRI of the cervical spine confirming pathology capable of causing compression or irritation of the vertebral artery (osteochondrosis, spondylosis, Kimmerle anomaly, instability, post-traumatic changes) [10, 28].
3. **Angiographic criterion** - results of ultrasound Doppler with functional tests, MR- or CT-angiography demonstrating impaired blood flow (decrease in linear blood flow velocity >50% during rotation, asymmetry >30%, hemodynamically significant stenosis, extravascular compression, or hypoplasia) [29, 34, 55].

Algorithm of VAS Diagnosis (Simplified Scheme)

The diagnostic process includes sequential stages: from suspicion of VAS (complaints and connection of symptoms with head movements) to neurological examination with provocative

tests (primarily De Kleyn), visualization of the cervical spine (X-ray/CT/MRI), assessment of hemodynamics (ultrasound Doppler with functional tests), and MRI of the brain to exclude other focal pathology. According to indications, MRA/CT angiography, TCDG, vestibular tests, audiometry, and cardiological examination are performed. The process is completed by differential diagnosis and assessment of compliance with all three diagnostic criteria, with determination of the cause, stage, and form of the disease [4, 26, 29, 34, 35].

Conclusion (Diagnosis)

Diagnosis of VAS requires an integral interpretation of clinical, imaging, and functional data in their dynamics. No single method is sufficient on its own. Of decisive importance are the clear connection of symptoms with head movements, ultrasound Doppler with functional tests as the main way to confirm the hemodynamic significance of compression, MRI/CT to verify the vertebrogenic cause and exclude other brain diseases, as well as thorough differential diagnosis. Only a comprehensive approach ensures accurate diagnosis, identification of causative factors, and selection of adequate treatment tactics [4, 12, 26, 29, 34, 35, 55].

Treatment of Vertebral Artery Syndrome (VAS)

Treatment of VAS is a complex, individual, and long-term process aimed at eliminating the cause of impaired blood flow, improving hemodynamics in the vertebrobasilar basin, relieving symptoms, and preventing severe complications (TIAs, stroke). The tactics are determined by the cause, stage of the disease, clinical picture, and examination results [6, 36].

I. Conservative Treatment (the main approach for the majority of patients) The foundation of therapy is lifestyle modification and elimination of provoking factors. An orthopedic pillow (medium height with a recess), correction of the workplace, regular breaks, and avoidance of sharp turns and tilting of the head are mandatory. It is necessary to quit smoking, control blood pressure, lipid profile, and body weight, and maintain moderate physical activity (walking, swimming).

Drug therapy includes vasoactive drugs (cinnarizine, vinpocetine, ginkgo biloba derivatives, piribedil), antiplatelet agents (acetylsalicylic acid, clopidogrel, dipyridamole), neuroprotectors and antioxidants (piracetam, glycine, mexidol). Symptomatic treatment provides for NSAIDs and muscle relaxants for pain, betahistine as the drug of choice for dizziness, vestibular suppressants (short courses), as well as agents for correction of autonomic and cochlear disorders [6, 36, 44].

Physiotherapy (electrophoresis, magnetotherapy, laser therapy, ultrasound, mud therapy, balneoprocedures) effectively complements the treatment [28, 50].

Therapeutic physical culture is a key component: it strengthens the deep neck muscles, improves mobility, and forms the correct motor stereotype using mainly isometric exercises. Classes should be regular, smooth, and painless [11, 45, 63].

Manual therapy and massage (soft techniques, post-isometric relaxation) are used with great caution only by qualified specialists after MRI. Additionally, acupuncture, PIR, and botulinum toxin therapy are used for persistent muscle spasm [9, 11].

II. Surgical Treatment Surgical intervention is indicated in case of ineffectiveness of conservative therapy within 6 months and the presence of hemodynamically significant compression, frequent TIAs, syncope, drop attacks, or progressive neurological deficit. The main methods are decompression of the vertebral artery (for Kimmerle anomaly, osteophytes), vascular reconstructions (stenting, shunting), and stabilizing operations on the cervical spine [41, 48, 58].

III. Treatment of Complications TIAs and ischemic stroke in the vertebrobasilar basin require emergency hospitalization, antiplatelet or anticoagulant therapy, statins, antihypertensive treatment, and early rehabilitation [6, 52].

IV. Rehabilitation Vestibular rehabilitation is of decisive importance for dizziness and unsteadiness. It includes habituation exercises, balance training, and visual stabilization, which significantly improves quality of life and reduces the risk of falls [45, 50, 63].

V. Prognosis and Prevention The prognosis depends on the cause, stage, and adherence to treatment. With timely comprehensive therapy, it is favorable in most cases of muscular-tonic and moderate degenerative forms. Prevention of exacerbations includes adherence to an orthopedic regimen, regular courses of drug support, exercise therapy, vestibular rehabilitation, and dynamic monitoring by a neurologist [45, 63].

The success of VAS treatment is determined by its complexity, individual approach, duration of therapy, and active patient participation. Treatment should be carried out under the supervision of a neurologist with the involvement, if necessary, of a vertebrologist, manual therapist, vascular surgeon, and exercise therapy specialist [6, 36].

Conclusion Vertebral artery syndrome (VAS) is one of the most common and clinically significant forms of vertebrobasilar insufficiency in the structure of neurological pathology. In recent years, there has been an increase in its prevalence due to population aging, physical inactivity, prolonged work with digital devices, and occupational overload of the cervical spine. The disease is multifactorial in nature, based on degenerative-dystrophic changes (cervical spondylosis), vascular disorders, and muscular-tonic syndromes [1, 2, 7, 14, 15].

The review systematizes modern data on the etiology, pathogenesis, classifications (etiopathogenetic, clinical, topical, by stages and mechanisms), diagnostic criteria according to N.V. Vereshchagin, and examination algorithms. The leading role of comprehensive diagnosis, including detailed history taking, provocative tests (De Kleyn test), neuroimaging (MRI, CT, MRA), and ultrasound Doppler with functional tests, is emphasized [4, 12, 26, 34, 35].

Treatment of VAS should be individual, complex, and long-term. The basis is conservative therapy: lifestyle modification, drug correction, exercise therapy, physiotherapy, and vestibular rehabilitation. Surgical intervention (vertebral artery decompression, stenting) is indicated in refractory forms and high risk of complications. Early neurorehabilitation and prevention of exacerbations are of great importance [6, 11, 36, 45, 50, 63].

Despite the achieved successes, issues of standardization of diagnostic criteria in accordance with ICD-11, development of prognostic models, and optimization of rehabilitation programs remain unresolved. Further prospective studies using biomarkers and functional imaging will increase the effectiveness of early diagnosis and personalized treatment of VAS [38, 44, 57, 61, 62].

Thus, vertebral artery syndrome requires close attention from neurologists, vertebrologists, and rehabilitation specialists. Timely detection, a comprehensive approach to therapy and prevention can significantly improve the quality of life of patients and reduce the risk of severe complications [5, 16, 19, 54, 58].

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