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TACTICS OF MANAGEMENT OF PATIENTS WITH PAZVANOCHNAYA ARTERY SYNDROME

Urinov Muso Boltaevich, Saidov Sukhrob Rustamovich
Bukhara State Medical Institute

Abstract:

The article summarizes the literature data on vertebral artery syndrome, its clinical manifestations, etiology and pathogenesis. Based on their own results, the possibilities of pathogenetic therapy of the disease with nicergoline are considered. 22 patients aged 21-71 years were treated with nicergoline (half of them were treated in a hospital, the rest on an outpatient basis). The duration of treatment ranged from 2 to 6 months. The positive effect of nicergoline on the most common clinical symptoms, such as headache, dizziness, as well as a persistent or sharp (crisis) increase in blood pressure, has been established. Long-term therapy with nicergoline showed a significant improvement in the quality of life of patients on the SF-36 scale. The effectiveness of nicergoline therapy has been established for any variant of the development of vertebral artery syndrome, i.e. regardless of the cause of this condition

Keywords: *vertebral artery syndrome, diagnosis, clinical symptoms, treatment.*

Introduction

An analysis of the literature data showed that currently there is no unambiguous definition and unified classification of vertebral artery syndrome (PA), this allows these diagnoses to determine various pathologies — posterior cervical sympathetic syndrome (Barre—Lieu syndrome, cervical migraine, spinal nerve lesion syndrome), vertebrogenic cervicocranialgia or to use a generalized term — vertebral-basilar insufficiency and/or chronic cerebral ischemia. In the review part of this article, an attempt is made to clarify the concept of the term and classification of PA syndrome. At the same time, new possibilities of pathogenetic therapy and improvement of the patient's quality of life are also considered.

The International Classification of Diseases of the 10th revision (ICD-10) indicates several diagnoses characterizing damage to the vertebral-basilar bed: M.47.0 — PA compression syndrome in spondylosis (PA syndrome); M.53.0 — cervical cranial syndrome (posterior cervical sympathetic

syndrome); G.45.0 - syndrome of the vertebral-basilar arterial system [18, 19, 27]. PA syndrome is usually diagnosed when a vertebrogenic variant of PA compression is assumed [26].

If the vertebrogenic effect is directly affected by PA, we are talking about a compression variant of PA syndrome, which is of key importance in the presence of atherosclerosis of the arteries of the vertebral basilar system, especially bilateral, when the possibility of collateral blood flow is practically excluded, and the syndrome of the vertebral basilar arterial system occurs. In this case, they speak of vertebral-basilar insufficiency and/or violation of cerebral circulation in the vertebral-basilar arterial system [4, 20, 22, 23]. If the fibers of the sympathetic plexus surrounding the PA are compressed, then we mean an irritative variant of the PA syndrome with the occurrence of vascular spasm not only in the PA directly subjected to compression, but also in the opposite PA and in the arteries of the overlying carotid basin. The irritative type of PA syndrome is usually observed in young patients who do not have a combined pathology of the vertebral-basilar bed. In people of the older age group, a mixed compression-irritative variant of PA syndrome is most often found. In addition, PA spasm may develop as a result of a reflex response to irritation of the afferent structures of the affected vertebral-motor segment of the cervical spine. In this case, PA syndrome is designated as reflex angiospastic, the degree of clinical manifestation of which almost always remains mild or moderate. However, it should be noted that in some cases it is a minor chronic irritation of the vertebral nerve and periarterial sympathetic plexus of the PA that leads to pronounced changes in the tone of brachiocephalic and cardiac vessels, dystrophic changes in the myocardium and neck muscles. This feature is important because many authors in their works focus on severe, easily detectable compression factors, underestimating the role of muscle and reflex mechanisms [5].

Thus, PA syndrome is a collective concept that combines a complex of cerebral, vascular, and vegetative syndromes resulting from damage to the sympathetic plexus of the PA, deformation of its wall, or changes in the lumen [6]. The prevalence of PA syndrome in degenerative-dystrophic manifestations of pathology of the cervical spine is 30.0—42.5% [11].

Historically, the first descriptions of the close relationship between the cervical spine, the PA and the sympathetic plexus surrounding it date back to 1925, when M. Wagge [15] described an unusual type of headache combined with visual, auditory, vestibular and autonomic disorders in patients with osteochondrosis and spondylosis of the cervical spine. Subsequently, his student Y. Lieou called this symptom complex posterior cervical sympathetic syndrome. W. Bartschi-Rochain, studying traumatic injuries of the cervical spine, used the term "cervical migraine" [16]. Note that this term is rarely used in Russian neurology, although it is used by many both foreign and domestic authors [3, 24, 25].

Currently, there are numerous additional methods of examining the patient (radiography of the cervical spine with functional tests, radiography of the craniovertebral junction, duplex scanning of the brachiocephalic arteries, arteries of the vilisium circle, magnetic resonance and X-ray computed tomography of the cervical spine, etc.), which help in diagnosing PA syndrome. However, despite the available possibilities, accurate diagnosis of PA syndrome is often difficult, since this condition does not always meet clear clinical and diagnostic criteria and requires long-term monitoring of the patient with the development of an individual plan for his examination and treatment.

The topographic and anatomical location of the PA in a narrow movable bone canal formed by transverse processes of the cervical vertebrae creates the possibility of its traumatization. PA penetrates into this channel through an opening in the transverse process of the CVI vertebra, less often of the CVI, CV or CIV vertebrae. In this channel, PA is accompanied by a thick sympathetic nerve plexus, which is associated with cervical sympathetic nodes, including VERTEBRAL ARTERY SYNDROME with a stellate node (cervical-thoracic node, gangl. stellatum), which provide innervation not only of the vertebral-basilar basin, but also give branches to the heart,

larynx, etc. In addition, numerous branches return from the sympathetic plexus to the vertebrae, joints and intervertebral discs. After passing through CI, the PA is located in the furrow on the posterior arch of the Atlas, where it is covered by almost only one lower oblique muscle of the head and then penetrates through the large occipital foramen into the cranial cavity. Given the described anatomical features of PA, it is easy to assume that even minor degenerative-dystrophic changes in the cervical spine can lead to the occurrence of PA syndrome. Hyperextension injuries of the cervical spine are of great importance in the formation of PA syndrome, especially with rotation of the cervical spine — "whiplash injuries" ("whiplash injury") with the appearance of a characteristic clinical picture of PA syndrome in both acute and long-term periods [8, 19]. Chronic irritation of the sympathetic plexus has been well studied in cervical spondylosis [27], uncovertebral arthrosis, posterolateral herniated discs, instability of the motor segments of the cervical spine, various anomalies of the craniovertebral junction, congenital blocks of the cervical spine, hypoplasia and/or congenital or acquired pathological tortuosity of the PA, after manual manipulations on the cervical spine, especially when there are pronounced degenerative changes and other pathology of the cervical spine [2, 10, 28].

It is known that the mediator of the sympathetic nervous system is norepinephrine, which is contained in the endings of sympathetic fibers. Norepinephrine is a precursor to adrenaline, but differs from it by a much more powerful vasoconstrictor and pressor action. Norepinephrine participates in the regulation of blood pressure and peripheral vascular resistance, has an activating effect on the reticular formation; it is the most important mediator of wakefulness. The effect of norepinephrine is associated with a predominant effect on alpha-adrenergic receptors. The cardiotropic effect of norepinephrine is associated with its stimulating effect on the β-adrenoreceptors of the heart [1, 9]. Prolonged violation of vascular innervation due to cervical osteochondrosis can lead to a persistent change in blood pressure, when even etiotropic therapy does not improve. This is due to the fact that a vicious circle arises: osteophytes of the cervical vertebrae cause compression of the PA and irritation of its sympathetic plexus. Under certain conditions, due to anatomical connections, branches of the internal carotid artery are also involved in the process, and irrigation exacerbates vascular spasm. As a result, ischemia and defective functioning of the hypothalamic region occur, which is aggravated by repercussive changes in the hypothalamus due to irritation of the sympathetic plexus of the PA [13, 14, 27].

The clinical manifestations of PA syndrome depend on the causes of this condition. Post-traumatic PA syndrome has a high degree of neurological symptoms and a vivid clinical picture. Chronic irritation of the surrounding sympathetic plexus, as mentioned above, leads to persistent tone of the brachiocephalic and cranial arteries and is manifested by cerebrovascular disorders of varying degrees. Concomitant diseases such as atherosclerosis of the vessels of the brachiocephalic arteries, brain, hypertension, coronary heart disease or a combination of them are also of great importance in the symptom complex of PA syndrome. It should be noted that patients with chronic vertebral-basilar insufficiency caused by vascular and extravascular factors also have episodes of PA syndrome, which is usually verified as an exacerbation of vertebral-basilar insufficiency. In this regard, for an accurate diagnosis, anamnestic data should be taken into account, especially events that immediately preceded the development of PA syndrome — discoordinated movements in everyday life (reaching for water, taking something from the top shelf, etc.) or during sports, prolonged stay in a static position (working with your head thrown up, reading in a position of deep flexion and / or rotation of the neck, hyperextension of the cervical spine during tooth traction), sleeping in an unusual place (at a party, on a newly purchased sofa), performing manual manipulations.

The most common clinical variants of PA syndrome are: posterior cervical sympathetic syndrome — a kind of symptom complex, regarded as a functional stage of PA syndrome [7]. It is characterized by headache in the cervical-occipital region of a pulsating or burning nature with

irradiation to the anterior parts of the head and the ocular region, accompanied, in some cases, by lacrimation, nasal congestion and other vegetative manifestations. Headache with this syndrome can be constant, especially in the mornings after sleeping on an uncomfortable pillow, when walking, shaking, and neck movements [11]; vestibulo-atactic syndrome — most often accompanies headache (subjective symptoms prevail: dizziness, feeling of body instability, darkening of the eyes, imbalance with nausea and vomiting, cardiovascular disorders [10, 12, 17]; cochleo-vestibular syndrome; ophthalmic syndrome; syndrome of autonomic disorders (up to severe hypothalamic crises).

In severe cases of the disease, transient circulatory disorders in the vertebral-basilar system, drop attacks, and syncopal conditions develop [4].

Summarizing the above, it is possible to identify the characteristic features of PA syndrome in terms of its difference from other vascular syndromes. PA syndrome develops as a result of vertebrogenic effects on the sympathetic nervous plexus of PA and/ or direct compression of PA and is manifested by characteristic clinical symptoms associated primarily with the release of norepinephrine into the systemic bloodstream, which, by binding to the alpha-adrenergic receptors of the vessels, has a systemic and peripheral vasospastic effect. The severity of symptoms varies from mild (headache, dizziness, slight rises in blood pressure) to significant (severe hypothalamic crises and/ or a sharp simultaneous increase in blood pressure) and depends on the etiopathogenetic factor that caused this condition.

This work was carried out taking into account existing ideas about the etiology and mechanism of development of PA syndrome and direct involvement in the development of symptoms of adrenergic mechanisms. Its purpose was to evaluate the effectiveness of the drug nicergoline (nicergoline) in patients with PA syndrome.

Nicergoline is a derivative of ergot and is currently used in more than 50 countries around the world [21]. Nicergoline contains an ergoline core and a bromine-substituted nicotinic acid residue, and exhibits an α-adrenoblocking effect [29]. It is the α-adrenoblocking effect that determines its pharmacotherapeutic effectiveness in PA syndrome. This effect significantly distinguishes nicergoline from other vascular drugs, the vasodilating effect of which is carried out using other mechanisms. The appointment of drugs that do not have an alpha-adrenoblocking effect has a symptomatic, but not pathogenetic orientation, gives good results, and can be shown in cases of poor nicergoline tolerance. However, it should be borne in mind that the effect of symptomatic therapy is less prolonged. The duration of therapy with nicergoline is determined by the degree of improvement in the patient's clinical condition and averages from 3 to 6-12 months.

Material and methods

We observed 42 patients, 16 men and 26 women aged 18 to 59 years (average — 42 years) with PA syndrome with or without concomitant pathology. The duration of the disease ranged from several months to 12 years; at the same time, 21 patients were treated in a day hospital and 21 were treated on an outpatient basis (in the latter case, the assessment of the condition of patients was carried out every 3-5 days). The total duration of follow-up of patients ranged from 2 to 6 months.

The doses of nicergoline in the treatment of patients in a day hospital were 4 mg per 100 ml of 0.9% NaCl solution intravenously drip 1 time per day for 12 days with further transfer to tablet administration of the drug — 10 mg 3 times a day. When treating patients on an outpatient basis, nicergoline was prescribed 10 mg 3 times a day.

The diagnostic algorithm included a standard neurological examination, an assessment of headache on the visual analog pain scale (VAS); an assessment of indicators characterizing a vertigo attack, in particular the frequency of seizures per day and their intensity on the Dizziness Handicap Inventory scale. The quality of life of patients was also assessed on the SF-36 scale.

YOURS was 10 cm long. The starting point of the line indicated the absence of pain — 0, followed by mild, moderate, severe, final, unbearable pain — 10. The patient was required to mark the pain level with a dot on this straight line. Pain is a subjective qualitative sign and has inaccuracies in its interpretation by patients. In this regard, the scale is divided into 6 degrees of pain, where 0 means no pain, 1-2 cm — mild pain, 3-4 cm — moderate, 5-7 cm — severe, 8-9 cm — terminal and 10 cm — unbearable pain.

According to the Dizziness Handicap Inventory dizziness scale, the assessment was carried out in the following gradations: I — dizziness attacks do not bother; II — minimal symptoms of dizziness; III — mild symptoms of dizziness when turning the head or standing up, which do not disrupt normal life and are not accompanied by vegetative disorders; IV — moderate symptoms of dizziness: dizziness attacks lasting up to 10 seconds, minimal restrictions on motor activity, possible vegetative disorders (nausea, vomiting, imbalance, headache); V — pronounced symptoms of dizziness: attacks of dizziness lasting up to 30 seconds, moderate restrictions on motor activity, vegetative disorders in most cases of dizziness (nausea, vomiting, imbalance, headache, visual impairment, weakness, falling, numbness in the extremities), anxiety, anxiety about possible loss of balance and falling; VI — significantly pronounced symptoms of dizziness: seizures lasting more than 30 seconds, always accompanied by vegetative disorders (nausea, vomiting, imbalance, headache, visual impairment, weakness, falling, numbness in the extremities), severe restriction of motor activity, fear of possible loss of balance and falling.

The effect of treatment was assessed in these degrees of gradation — by how many degrees (gradations) the patient's clinical condition improved in terms of reducing the severity of dizziness and headache as qualitative signs in accordance with the scale of dizziness and headache given above.

Statistical processing of the results was carried out using the Statistica 6.0 software package. To assess the significance of qualitative signs (headache and dizziness), the Wilcoxon sign rank criterion was applied.

Результаты и обсуждение

Nicergoline therapy made it possible to achieve a highly reliable reduction in the frequency and intensity of dizziness attacks ($p<0.001$), as well as the severity of headache ($p<0.001$). So, for example, reflecting episodes of dizziness, it follows that before treatment 6 patients had significantly pronounced symptoms of dizziness (grade VI on the vertigo scale), after therapy with nicergoline, they were not disturbed by dizziness attacks, i.e. the frequency and severity of dizziness in these patients decreased by 5 gradations. Comparing the quality of life of patients using the quality of life scale (SF-36) before and after treatment with nicergoline revealed a significant improvement in the physical (before treatment — 28.3±7.9, after treatment — 53.1±3.6 points, $p<0.001$) and psychological components of health (before treatment — 29.7±12.3, after treatment — 57.4±6.9 points, $p<0.001$).

After inpatient treatment, pain in the cervical spine bothered for a long time, the above-described paroxysms began to appear with a frequency of 1 time per month. I turned to a chiropractor for help. After manual manipulations, I felt some improvement. He was observed for a long time by a therapist, a cardiologist, and was examined for hypertension. No data were obtained for the symptomatic nature of hypertension. The prescribed antihypertensive therapy did not bring relief: the patient maintained simultaneous sharp rises in blood pressure during headache attacks; the patient refused to take antihypertensive drugs.

During the examination of the patient in the clinic, pronounced changes in the cervical spine were revealed — osteochondrosis, uncovertebral arthrosis, osteophytes of vertebral bodies with maximum manifestations at the level of CIV—CVI, straightening of cervical lordosis, herniated intervertebral discs at the level of CV—CVI, CV—CVII. Based on clinical and anamnestic data and

the results of an additional examination, an irritative variant of PA syndrome (posterior cervical sympathetic syndrome) was diagnosed. As a basic therapy, additional treatment with nicergoline at a dose of 10 mg 3 times a day was prescribed, as well as rehabilitation techniques. The effectiveness of therapy was revealed already on the 3rd day of taking the drug: paroxysmal conditions completely disappeared, blood pressure normalized and work capacity was restored.

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