

ESTIMATION OF EFFICIENCY OF COMPLEX THERAPY OF PROGRESSING GLAUCOMA NEUROPATHY

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ОЦЕНКА ЭФФЕКТИВНОСТИ КОМПЛЕКСНОЙ ТЕРАПИИ ПРОГРЕССИРУЮЩЕЙ ГЛАУКОМНОЙ НЕЙРОПАТИИ

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РИВОЖЛАНУВЧИ ГЛАУКОМАТОЗ НЕЙРОПАТИЯНИ КОМПЛЕКС ДАВОЛАШНИНГ САМАРАДОРЛИГИНИ БАХОЛАШ

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Оценка терапевтической эффективности глиатилина для стабилизации зрительных функций после комплексного лечения больных прогрессирующей глаукомно оптической нейропатии с «нормализованным» давлением. **Глиатилин** - ноотропный препарат, холиномиметик центрального действия с преимущественным влиянием на ЦНС. Включение глиатилина в комплекс лечебных мероприятий, направленных на поддержание зрительных функций у больных нестабилизированной глаукомой, направленных на различные звенья патогенеза глаукомной оптической нейропатии позволяют добиться стабилизации процесса у 89% больных далеко зашедшей нестабилизированной глаукомой в течение 6 месяцев.

Ключевые слова - первичная глаукома, глаукомно оптическая нейропатия, глиатилин, ноотроп.

«Нормаллаштирилган» босимли, ривожланувчи глаукоматоз оптик нейропатияли беморларни комплекс даволашдан кейин, кўрув фаолиятларини барқарорлаштириш учун, глиатилиннинг терапевтик самарадорлигини баҳолаш. Глиатилин ноотроп дори воситаси бўлиб, марказий асаб тизимига бирламчи таъсир кўрсатадиган марказий холиномиметик воситадир.

Глиатилиннинг барқарорлашмаган глаукома билан оғриган беморларда кўрув фаолиятларини сақлаб туришга қаратилган, глаукоматоз оптик нейропатия патогенезида, турли хил таъсир этувчи, терапевтик чора-тадбирлар мажмуига киритилиши, оптик давомиди узок, муддатли барқарор бўлмаган глаукомали беморларнинг 89 фоизиди, жараёни барқарорлаштиришга имкон беради.

Калит сўзлар - бирламчи глаукома, глаукома-оптик нейропатия, глиатилин, ноотроп.

PPrimary glaucoma, despite advances in ophthalmology still occupies one of the first places among the causes of blindness throughout the world. Ophthalmologists are well aware that even with the achievement of persistent IOP compensation by medication or surgery, every 5th patient continues to decompose visual functions [1- 3,5]. In this regard, the problem of treating glaucoma optic neuropathy is very relevant.

Glaucoma optical neuropathy fGONJ is usually considered from the standpoint of mechanical, vascular and metabolic theories, including numerous risk factors that increase the likelihood of progression of glaucoma lesions. Determining the primacy of a factor is always debatable. Only one thing is obvious: in the case of achieving an individual level of intraocular pressure (IOP) and at the same time marked progression of GON, it is necessary to identify other, most likely factors of influence.

Given the multifactorial progression of GON progression, ophthalmologists usually recommend complex therapy, prescribing drugs of various pharmacological groups [6-8]. This is often done when they plan to study the effectiveness and safety of one of the drugs included in complex therapy.

Considering metabolic disorders, among which the leading place is occupied by excitotoxic damage to the third retinal neuron and activation of free radical processes in the retina and optic nerve [1,4], we considered it expedient to include drugs and treatment methods that improve, on the one hand, metabolism, on the other hand, neutralize the negative influence of a number of factors and, on the third hand, stimulate the activity of retinal neurons and restore the conductivity of nerve fibers.

Gliatilin is a nootropic drug, a central cholinomimetic with a primary effect on the central nervous system. Choline is released from the active substance in the brain; choline is involved in the biosynthesis of acetylcholine (one of the main mediators of nervous excitation). Alfoscerate is

biotransformed to glycerophosphate, which is a precursor of phospholipids.

Acetylcholine improves transmission of nerve impulses, and glycerophosphate is involved in the synthesis of phosphatidylcholine (membrane phospholipid), resulting in improved membrane elasticity and receptor function. Gliatilin increases cerebral blood flow, enhances metabolic processes and activates the structure of the reticular formation of the brain, restores consciousness in traumatic brain damage. It has a preventive and corrective effect on factors of involutional psycho-organic syndrome, such as a change in the phospholipid composition of neuronal membranes and a decrease in cholinergic activity. Thus, pharmacodynamic studies have shown that gliatilin acts on synaptic, including cholinergic, transmission of a nerve impulse (neurotransmission), plasticity of a neural membrane, and receptor function.

Despite the existing difficulties in evaluating the effectiveness of neuroprotective therapy due to the lack of abso

lutely reliable criteria for a number of structural and functional indicators, such an assessment is still possible.

The aim of this work is to evaluate the therapeutic efficacy of gliatilin for stabilizing visual functions after the complex treatment of patients with progressive GON with "normalized" pressure.

Material and methods

We studied the effectiveness of treatment in two groups, the proposed method in 52 patients (61 eyes) with primary open-angle glaucoma in the advanced stage with compensated intraocular pressure. Patients of both groups were comparable in age, concomitant somatic pathology, the severity of the glaucoma process, their average age was 71.3 ± 1.6 years. In all patients, therapists diagnosed systemic atherosclerosis with a predominant damage to the vessels of the brain and cerebrovascular insufficiency.

The analysis of ophthalmostatus indices showed that in both groups the majority were patients with advanced stages of glaucoma: 71.3% and 73.6%, respectively. In the 2nd group, patients prevailed in which IOP normalization was achieved surgically: 79.3% versus 51.7% in the 1st group. Therefore, in the latter there were more patients who needed local antihypertensive therapy to maintain ophthalmotonus within the target pressure. Nevertheless, despite a steady level of IOP in the range of 15-17 mm Hg, negative dynamics of visual functions was noted in all cases, which served as the basis for the course of stabilizing therapy.

When conducting complex treatment of patients, their general somatic state was also taken into account. The course of neuroprotective therapy included drugs of various pharmacological groups acting on different pathogenetic links. All patients received Mexidol 100 mg intramuscularly 1 time per day for 14 days, and patients of the 1st group, in addition, were injected with gliatilin 1000 mg/4 ml intravenously in an amount of 10 injections, then continued the course of taking this drug by mouth 1 capsule 2 times a day for 3 months.

A comprehensive ophthalmological examination was carried out before, after treatment, after 3 and 6 months. The following methods were used to assess visual functions: visometry, perimetry, determination of the critical-frequency of flicker fusion, eye rheography. Along with this, a study was made of the electrosensitivity and electrolability of the optic nerve and retina, and the registration of visually evoked cortical potentials (VECP).

The state of the visual fields was evaluated in several ways. Static perimetry was performed using a Humphrey Visual Field Analyzer II (HFA II) 750i (Germany). Depending on the initial visual acuity and the degree of visual impairment, a screening or threshold study program was used. When assessing the central field of view (CTO), all patients underwent correction of visual acuity near. Screening was performed using the FF- 120 Screening program using a three-zone strategy. The threshold program for the study of the visual field included the application of tests Central 30-2 in the study of the central lens (within 30° from the point of fixation of the gaze] and Peripheral 60-2 in the assessment of the peripheral field of vision - the primary brain (from 30° to 60°). At the same time, we analyzed the threshold foveolar photosensitivity, the sum of decibel threshold values in each quadrant over the entire

field of view, mean deviation (MD] and standard deviation (PSD] deviations calculated automatically by the device taking into account its own database.

The criteria for evaluating the effectiveness of neuroprotective therapy are not sufficiently informative, and from the point of view of practical ophthalmology, the study of visual functions - perimetry - remains the most accessible.

Results and discussion

During the observation period in a hospital, during which patients received drugs, in no case were adverse events recorded. The IOP level was also normalized during the entire observation period and was at a level not exceeding 15 mm Hg ($p > 0.05$ compared with the initial data).

One of the criteria for evaluating functionality is central visual acuity. Neuroprotective therapy, as a rule, does not affect this indicator, however, mention of it is important as indirect evidence of the dynamics of the process.

In our case, central visual acuity remained stable. Some improvement in vision was noted in some patients in both groups. Visual acuity indicators increased from 0.32 ± 0.06 to 0.47 ± 0.07 [$p < 0.05$].

One of the objective criteria for assessing the effectiveness of neuroprotective therapy, to a certain extent, can be considered a study of the visual field. The indicators taken into account when assessing changes in visual functions, we considered CPL, foveolar and total photosensitivity, PPZ, indicators MD and PSD.

The study was conducted before the start of a course of drug therapy and 3, 6 months after it. All average indicators tended to improve, especially for the central and peripheral fields of vision. Moreover, in the group of patients receiving gliatilin, this trend was more significant. This is all the more important since the initial data of both groups were comparable. The boundaries of peripheral vision (the sum of degrees along 8 meridians] from $307 \pm 31^\circ$ to 365 ± 44 ($p < 0.05$), CFSM from 23 ± 6.0 to 29 ± 7.0 ($p < 0.05$) after treatment. This amounted to 47%, 19%, 26% of the initial level, respectively.

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Against the background of an increase in visual functions, we noted an improvement in hemodynamic and electrophysiological parameters.

The reographic coefficient increased from 1.52 ± 0.07 to 2.07 ± 0.14 ($p < 0.05$), which amounted to 36% of the initial indicator. The decrease in the threshold of electric

phosphene was 21.1 pA after treatment and 24.2 pA after 6 months of follow-up.

A significant increase in the index of electrolability of the optic nerve was established by an average of 2.3 Hz after treatment. After half a year of dynamic observation, the indicator is 3.5 Hz, which is 13% of the initial level, but this difference is not statistically significant.

As a result of the treatment, a positive dynamics of the state of visual functions was revealed according to the VEP study. The amplitude of the P 100 component increased from 11.7 ± 4.7 to 14.3 ± 5.1 pV.

We are inclined to believe that a more pronounced therapeutic effect in patients of the 1st group is due to the action of gliatilin. This assumption is confirmed by the observation of a large number of patients who periodically receive similar therapy at the institute.

Conclusion

Thus, the inclusion of gliatilin in the complex of therapeutic measures aimed at maintaining visual functions in patients with unstabilized glaucoma, aimed at various links in the pathogenesis of glaucoma optical neuropathy, makes it possible to achieve stabilization of the process in 89% of patients with far-reaching unstabilized glaucoma for 6 months.

Thus, the inclusion of gliatilin in the complex of therapeutic measures aimed at maintaining visual functions in patients with unstabilized glaucoma, aimed at various links in the pathogenesis of glaucoma optical neuropathy, makes it possible to achieve stabilization of the process in 89% of patients with far-reaching unstabilized glaucoma for 6 months.

The frequency of the course of stabilizing therapy depends on the effectiveness of the previous therapy and the clinical manifestation of the glaucoma process.

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Key words: primary glaucoma, glaucoma-optical neuropathy, gliatilin, nootrope.

