Pathogenetic aspect of blepharospasm according to the autonomic resonance test S.L. Chepurnaya (MBLPU GKB No. 1, Novokuznetsk, Russia)

Introduction

Blepharospasm is a form of cranial muscular dystonia, which consists in overactive circular muscle of the eye. This disease does not lead to the death of the patient, however, due to severe visual impairment, it often causes social maladjustment, difficulties in establishing interpersonal contacts, in professional activity and later difficulties in self-service, which gives the problem of blepharospasm an important medical and social sound [1, 7] ...

The etiology of the disease has not been finally established, which can be explained by the absence of specific specific pathomorphological, biochemical and electrophysiological markers. In pathological studies, according to Lys A.J., findings of two types were found: changes in the form of a distinct mosaic pattern in the dorsal half of the caudate nucleus and shell, or the absence of any changes at all [6].

The data given by E. Kandel and S. Voytyna [5, 7] indicate that the most pronounced changes take place in the globus pallidus, and the process is bilateral in nature. These changes are equally expressed throughout the above formations and relate primarily to astroglia and nerve cells. In the neurons of the globus pallidus, the phenomena of tigrolysis and the death of nerve cells are found [5, 6]. Since there is no pathogenetic and etiotropic drug therapy and data on the etiology and course of individual forms of blepharospasm, each individual case studied complements the picture of the pathogenesis of blepharospasm.

Materials and research methods

A patient with blepharospasm was examined using the ART method according to A.A. Hovsepyan [2, 3, 4].

Neurological examination [8, 9] revealed complaints of inability to open their eyes for more than 1 min, general weakness, severe weakness in the arms and legs, and increased drowsiness. At the time of wakefulness, irritability, rapid mood swings, and tearfulness were noted. The assessment of one's own actions is violated.

Anamnesis of the disease: fell ill 6 months ago, when for no apparent reason there was a twitching of the eyelids, then short-term involuntary shutting of the eyelids was added, increasing in intensity and duration, vascular, nootropic, vitamin therapy, acupuncture were performed, without effect. Involuntary spasm of the circular muscles of the eye, the muscles lifting the corner of the mouth, the zygomatic muscles progressed to the degree of the state of constantly closed eyes, the inability to open them on their own. The patient needs outside care. In neurological status: cannot keep eyes open in the supine position for 1 minute, tendon reflexes are revived, right above. Abdominal reflexes are preserved. Decrease in strength in the right limbs up to 4 points. Sensitivity is preserved. Pathological signs are outlined, more on the right. Adiadochokinesis on both sides. She is unstable in the Romberg position. Conducts coordination tests with dysmetry. Emotional lability. According to neuropsychological research according to Luria, mild cognitive impairments are revealed. The patient gets tired from the minimal load in the form of walking for 3-5 minutes.

Research results

The release mechanism of the mediator acetylcholine is discrete, quantum in nature. A feature of acetylcholine as a mediator is its rapid destruction after release from presynaptic endings using the enzyme acetylcholinesterase. Skeletal muscle motor neurons, spinal cord neurons, preganglionic neurons of intramural and extramural ganglia are cholinergic. In addition, they are found in the reticular formation of the midbrain, cerebellum, and basal ganglia. When examining a patient with a severe form of blepharospasm, an organ preparation of facial muscles revealed a change in the immune system in the form of depletion of cellular and humoral immunity, the presence of a viral infection. Excess acetylcholine, lack of ACTH, corticosteroids, corticotropin up to grade 6. Psychological load 3, 6, 7, 8 degrees, blockade of receptors. This indicates the presence of antibodies to anticholinesterase. The process of rapid destruction of acetylcholine is disrupted. An excess of acetylcholine arises in the synapses of the motor neurons of the facial muscles, which accumulates and causes their continuous contraction. In the cells of the reticular formation of the midbrain, cerebellum, basal ganglia, antibodies to acetylcholinesterase cause disruption of the neuronal activity of these structures and interneuronal connections in the brain. This is confirmed by MRI of the brain, which revealed multiple foci of demyelination in the hippocampus, basal ganglia, cortex, frontal, temporal lobes, as well as the presence of increased sleepiness, cognitive impairment, irritability, tearfulness, rapid mood swings, decreased criticism, initiative, volitional stimulus, social maladjustment. This indicates the presence of antibodies to anticholinesterase. The process of rapid destruction of acetylcholine is disrupted. An excess of acetylcholine arises in the synapses of the motor neurons of the facial muscles, which accumulates and causes their continuous contraction. In the cells of the reticular formation of the midbrain, cerebellum, basal ganglia, antibodies to acetylcholinesterase cause disruption of the neuronal activity of these structures and interneuronal connections in the brain. This is confirmed by MRI of the brain, which revealed multiple foci of demyelination in the hippocampus, basal ganglia, cortex, frontal, temporal lobes, as well as the presence of increased sleepiness, cognitive impairment, irritability, tearfulness, rapid mood swings, decreased criticism, initiative, volitional stimulus, social maladjustment. This indicates the presence of antibodies to anticholinesterase. The process of rapid destruction of acetylcholine is disrupted. An excess of acetylcholine arises in the synapses of the motor neurons of the facial muscles, which accumulates and causes their continuous contraction. In the cells of the reticular formation of the midbrain, cerebellum, basal ganglia, antibodies to acetylcholinesterase cause disruption of the neuronal activity of these structures and interneuronal connections in the brain. This is confirmed by MRI of the brain, which revealed multiple foci of demyelination in the hippocampus, basal ganglia, cortex, frontal, temporal lobes, as well as the presence of increased sleepiness, cognitive impairment, irritability, tearfulness, rapid mood swings, decreased criticism, initiative, volitional stimulus, social maladjustment. The process of rapid destruction of acetylcholine is disrupted. An excess of acetylcholine arises in the synapses of the motor neurons of the facial muscles, which accumulates and causes their continuous contraction. In the cells of the reticular formation of the midbrain, cerebellum, basal ganglia, antibodies to acetylcholinesterase cause disruption of the neuronal activity of these structures and interneuronal connections in the brain. This is confirmed by MRI of the brain, which revealed multiple foci of demyelination in the hippocampus, basal ganglia, cortex, frontal, temporal lobes, as well as the presence of increased sleepiness, cognitive impairment, irritability, tearfulness, rapid mood swings, decreased criticism, initiative, volitional stimulus, social maladjustment. The process of rapid destruction of acetylcholine is disrupted. An excess of acetylcholine arises in the synapses of the motor neurons of the facial muscles, which accumulates and causes their continuous contraction. In the cells of the reticular formation of the midbrain, cerebellum, basal ganglia, antibodies to acetylcholinesterase cause disruption of the neuronal activity of these structures

When testing the organopreparation of the Adrenal glands, a violation of the immune system was revealed in the form of depletion in the cell and tension in the humoral type, a lack of corticosteroids, and a viral infection. When examining the organopreparation, the liver revealed changes similar to those in the adrenal glands, which also indicated an autoimmune process. The clinic revealed a pronounced asthenic syndrome. Additional examinations confirmed liver damage in the form of hepatitis, confirmed by ultrasound examination, an increase in the level of ALT, AST in the blood.

All indicators: complaints, symptoms detected during clinical examination, the course of the disease indicate a systemic lesion of an autoimmune nature. Taking into account adrenal insufficiency, the presence of an autoimmune process, a trial therapy with Dexamethasone was carried out according to the scheme.

As a result, the degree of blepharospasm, asthenic syndrome decreased.

conclusions

The ART method allows the most accurate identification of pathogenetic changes occurring in the body as a whole and in individual organs, to determine the etiology in difficult cases in the absence of specific specific

pathomorphological, biochemical and electrophysiological markers, and to select bioresonance and drug therapy.

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