CLINICAL CASE OF ISAACS SYNDROME

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Introduction. Isaacs Syndrome (constant muscle fiber activity syndrome) is a rare autoimmune disease first described by N. Isaacs in 1961. There is no information about the frequency of Isaacs Syndrome (SI), since there are isolated publications in the literature devoted to the clinical or electrophysiological features of the disease in single patients. The main pathogenetic mechanism of SI development is the production of autoantibodies against potassium channels located in the distal parts of motor nerves or nerve terminals. As a result of potassium channel blockade, increased excitability of peripheral nerves is observed [3, p.354-356].

Purpose: to analyze the clinical case of Isaacs syndrome.

Materials and methods of clinical case. Patient P., 46 years old, an engineer, was admitted to the neurological department with complaints of difficulty in voluntary movements in the hands, more in the left, difficulty reading and changing gait. He was ill for about two years, when after physical exertion he began to notice a feeling of stiffness in his hands, could not perform movements that required accuracy.

After rest, these phenomena passed. In recent months, after intense physical exertion, the condition has significantly worsened: stiffness in the hands has increased, repeated movements in them have become slow and clumsy. At the same time, I noticed that when reading, it is difficult to look at a new line after reading the previous one. Among the previously transferred diseases, he notes chronic tonsillitis and myocardial infarction (7 years ago). Family and hereditary history is not burdened [1, p. 27-35].

Results. Upon admission, the patient's condition is satisfactory, correct build, dark skin. Blood pressure 140/100 mm Hg. St., pulse 72 beats / min, rhythmic, heart sounds muffled.

Neurological status: conscious, oriented, adequate, memory is not impaired. The movements of the eyeballs are devoid of smoothness, spasmodic. Active extension of the fingers of the hands is difficult, the pace of movements is slow, each subsequent movement has a smaller amplitude, after 4-5 movements, flexor contractures of the hands appear, mainly on the left. Foot movements are also difficult, but to a lesser extent. The muscles of the forearm, hands, shins and feet are dense to the Touch, increased muscle tone does not decrease during sleep. Muscle strength in the hands is reduced to 4 points. Fascicular twitches are noted in the muscles of the shoulder girdle. When a hammer hits the muscles of the distal extremities, a myotonic reaction occurs in the form of a roller. Tendon-periosteal reflexes are high, S>D, there are no pathological reflexes. Sensitivity is not impaired. Performs coordination tests satisfactorily. The gait is devoid of smoothness, the movements are clumsy, slow, the feet are hardly lifted off the floor.

Examination: general blood and urine tests without detected pathology. Needle EMG from the muscles of the upper and lower extremities: fasciculation potentials, repeated discharges of motor units and neuromyotonic discharges are recorded at rest. The duration and amplitude of motor unit potentials are within the normal range. With an arbitrary maximum reduction, the EMG interference type is recorded, larger on the left.

After the appointment of finlepsin (800 mg / day) and diacarb (75 mg / day), the patient's condition improved: stiffness in the hands and feet decreased, but with repeated repetitions of movements, there was a slight inconvenience in the hands, which did not interfere with self-care. During follow-up examinations after 6 and 12 months, he took finlepsin and diacarb, continued to work [2, p.67-81].

Conclusion. In the presented observation, the patient had a classic Isaacs triad: neuromyotonia, fasciculations, and muscle stiffness. The diagnosis was confirmed by the results of needle EMG. A special feature of the disease was the predominant and

asymmetric damage to the muscles of the hands, the involvement of the external muscles of the eyes in the process, and the preservation of deep reflexes in the limbs in the developed stage of the disease. Positive results of treatment with anticonvulsants were noted.

LIST OF LITERATURE:

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