Treating Guillain-Barre syndrome with corticosteroids (clinical case)

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Introduction. Guillain-Barre syndrome (GBS) is an acute autoimmune inflammatory polyradiculopathy, manifested with flaccid paresis, sensory disturbances, and autonomic disorders. GBS occurs worldwide, in people of different ages. The average age of Guillain-Barre syndrome occurrence is 40 years. More common in men. In world literature, there are described cases of GBS of a three-week newborn, and a 95- year-old man. Some authors distinguish two ages in which GBS peaks (20-29 years and 50 years), when it is diagnosed most often. On average, each year 1-2 people are afflicted with Guillain-Barre syndrome per 100 000. The disease often occurs irregularly. However, over the last 70 years outbreaks were observed, in which Guillain-Barre syndrome suddenly evolved from several tens to hundreds of people in various countries around the world - from China to Peru. Currently in the US and in Europe there is a tendency for increase in the number of patients with Guillain-Barre syndrome. One of the controversial components of GBS is the treatment, which includes plasmapheresis and administration of IgG. Admittedly this treatment is highly expensive and many regional hospitals are not able to carry it out. Many authors do not trust hormonal treatment, considering it questionable.

In this regard, we present a detailed description of our own clinical observations of a case with Guillain-Barre syndrome.

Anamnesis morbi. Patient P. (53 years) considers himself sick for about two days, when there was a weakness and pain in the left leg. He didn't seek medical help. There was alcohol consumption the previous day. During these two days the complaints described above increased and on admission the patient could not walk on his own. Was taken to the hospital by ambulance. In the emergency room was examined by a neurologist, who made a preliminary diagnosis of GBS, he was further examined by an internalist and maxillofacial surgeon, comorbid diseases - ulcerative glossitis. He was hospitalized in the neurological department for further examination and treatment.

Anamnesis vitae. Past illnesses - Denies having colds, tuberculosis, hepatitis A, viral hepatitis, HIV, contact with infectious disease patients (including measles). Bad habits: about once a week drinking small amounts of alcohol. Information on vaccination against measles was not provided. In 1977 had a surgery because of a penetrating wound to the chest. In 1978 there was a fracture of the left femur. During the year, there was a rise in blood pressure to 160 mm.Hg. isn't treated with antihypertensive drugs, wasn't tested.

Status objectivus. General condition of the patient is relatively satisfactory. Consciousness is clear, normal nutritional intake. Skin and visible mucosa of physiological color. There is a number of ulceritive elements on the lateral surface of the tongue. BP 150/90 mm.Hg. Pulse 85 per minute. Breathing vesicular, no wheezing. Abdomen soft, painless. At rest, shortness of wasn't observed. Physiological functions are normal.

Neurological status: CN - orbits D = S, pupils D = S, normal diameter. Photoreaction lively, D=S. Weakness of convergence on both sides. Symmetrical face, tongue at the midline. Pharyngeal reflex present. Axial signs ±. Tendon reflexes on hands D = S, somewhat reduced. Tendon reflexes on feet: knee D = S sharply reduced, Achilles tendon D = S sharply reduced. Plantar pathological signs - negative. Muscle strength - normal. Muscle tone - reduced. Sensory Disorders:

hypoesthesia of the lower extremities. Meningeal signs: stiff neck Kernig's sign, Brudzinski sign (upper, middle, lower) - not present. Coordination tests: finger-nose test with intention, heel-shin test with mild ataxia. Romberg's test wasn't performed. Pelvic disorders not present. Tension signs: not present. During his hospitalization, the patient's condition deteriorated. Weakness in the limbs and neck muscles was increased. Three days later, the patient was transferred to the intensive care unit.

After hospitalization the patient deteriorated. During the first three days, tetraparesis was observed. Power in the limbs is 2-3 points. Respiratory disorders weren't observed. Because of the increasing neurological symptoms in the form of increased tetraparesis. The patient was transferred to the intensive care unit. Within three days, sharp improvement detected. The patient voluntarily left the hospital, after which he was discharged for violation of hospital policy.

Presented clinical case is an example of an acute inflammatory autoimmune polyneuropathy. Admittedly the treatment of GBS is currently debatable. Taking into account that this is an autoimmune disease, we believe that the use of glucocorticoids in GBS is justified, as demonstrated by this clinical case.

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