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ORIGIN PATHOGENESIS OF HASHIMOTO 'S ENCEPHALOPATHY DISEASE

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Annotation: Hashimoto's encephalopathy, also known as steroid-responsive encephalopathy associated with autoimmune thyroiditis (SREAT), is a neurological condition characterized by encephalopathy, thyroid autoimmunity, and good clinical response to corticosteroids. It is associated with Hashimoto's thyroiditis, and was first described in 1966. It is sometimes referred to as a neuroendocrine disorder, although the condition's relationship to the endocrine system is widely disputed. It is recognized as a rare disease by the NIH Genetic and Rare Diseases Information Center . Up to 2005, almost 200 case reports of this disease were published. Between 1990 and 2000, 43 cases were published. Since that time, research has expanded and numerous cases are being reported by scientists around the world, suggesting that this rare condition is likely to have been significantly undiagnosed in the past. Over 100 scientific articles on Hashimoto's encephalopathy were published between 2000 and 2013.

Key words: Hashimoto , blood vassels, autoimmunity.

Hashimoto's encephalopathy (EH) is a rare neuroendocrine syndrome and is considered an autoimmune inflammatory brain lesion. In the world literature, this disease is known as steroid-reactive encephalopathy associated with autoimmune thyroiditis (steroid responsive encephalopathy associated with autoimmune thyroiditis (SREAT)), in which corticosteroid therapy in most cases has a significant positive effect.

The first descriptions of the syndrome appeared in the late 40s of the last century in reports of "myxedematous insanity", which was mistakenly associated with metabolic brain damage in hypothyroidism. Only a few decades later it was established that encephalopathy is not a neurological complication of hypothyroidism or thyroiditis Hashimoto's disease, and a kind of "parallel" disorder, having, like thyroiditis, an autoimmune nature.

Hashimoto's encephalopathy is a rare neuroendocrine syndrome and is considered an autoimmune inflammatory brain lesion associated with a high titer of antithyroid antibodies. The disease has an acute or subacute onset, manifests itself with intermittent tremor, myoclonus, epileptic seizures, ataxia, mental disorders and is characterized by a progressive or undulating course with the presence of spontaneous remissions. The drug of choice in the treatment of the disease are glucocorticosteroids.

Currently, EH is considered as encephalopathy with acute or subacute onset, manifested by intermittent tremor, myoclonia, epileptic seizures, ataxia, mental disorders, and characterized by a progressive or undulating course with the presence of spontaneous remissions, during which causes complete or partial reversibility of neurological and mental disorders. At the same time, vascular, infectious, tumor and other causes of brain damage are excluded.

According to the literature, the frequency of occurrence EH is 2.1 per 100,000 population. The average age of onset of the disease is about 44 years, with a fifth of all cases occurring in people younger 18 years old. Women suffer 4-5 times more often than men. The duration of the disease varies from 2 to 25 years. The variety of clinical manifestations of the disease is characteristic, imitating the picture of various neurological and psychiatric disorders that do not depend on the age factor. Therefore, due to non-specific symptoms and rarity, EH is hardly

recognized. Neurological symptoms most often debut with tremor, transient aphasia, epileptic seizures (Table.1). [2-4,13].

The detailed picture of EH usually develops quite quickly within 1-7 days and manifests itself with neurological symptoms in various combinations. Standard examination of cerebrospinal fluid (CSF) in most cases reveals normal cellular composition with an increase in protein content, the level of which reflects the activity of the process [6,7] Along the course of the disease, there are: progressive-remitting type (observed in most cases), occurs with characteristic stroke—like episodes with transient symptoms - transient aphasia, pronounced ataxia when walking, myoclonic hyperkinesis, etc.; diffuse progressive type, which is characterized by a progressive course with fluctuations in random order. The manifestation of the disease often begins with mental disorders followed by rapidly increasing dementia. In this type , tremor, myoclonus, epileptic seizures are often present in the clinical picture (in 20% of cases , epileptic status develops), stupor or coma often develops [5]. Frontal intermittent rhythmic delta activity (FIRDA),three-phase waves, regional deceleration, epileptiform activity, photoparoxysmal and photomiogenic reactions are often recorded on the EEG [1]. In neuroimaging studies, local nonspecific changes in subcortical white matter and cerebral atrophy are detected in half of the cases [2,3,5,8].

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