Syndrome of Amyotrophic Lateral Sclerosis at Vilyui Encephalomyelitis

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ABSTRACT

The review presents clinical polymorphism of Vilyui encephalomyelitis, variability and characteristics of the disease, when it is accompanied by the syndrome of amyotrophic lateral sclerosis. Diagnostic criteria and work-up in the differential diagnosis of Vilyui encephalomyelitis and classic amyotrophic lateral sclerosis are described.

Keywords: Vilyui encephalomyelitis (VEM), amyotrophic lateral sclerosis (ALS), a clinical polymorphism, bulbar palsy, pseudobulbar palsy.

Vilyui encephalomyelitis (VEM) is a severe progressive inflammatory disease of the Central Nervous System, with diffuse panencephalitis spreading of the pathological process, with often fatal outcome within a few months to 5-6 years or profound disability of the patient and affects mainly the aboriginal population of Yakutia.

The disease lies in the necrotic and inflammation processes, mainly in the gray matter of the brain. Pathologic studies the necrotic and inflammatory changes of the meninges or their consequences were detected in all patients with acute, subacute and chronic VEM. Micronecrotic lesions and inflammatory response to these lesions are widely spreading in the cerebral cortex, basal ganglia, cerebellum and brainstem. The inflammatory reaction manifested acutely with acute micronecrotic brain tissue lesions and continuing during the prolonged subacute phase are distinguishing features of VEM, but not short like usually in acute viral encephalitis [1, 2, 22].

VEM problem remains relevant to this day on the background extinction VEM epidemic that began in the 50th of the last century in Viliuisk areas [3, 13, 19]. But significant reduction in incidence in recent years does not guarantee that the epidemic Viliuisk encephalomyelitis will not flare up with renewed vigour, while still remain unresolved until the end of the etiology and pathogenesis of the disease and consequently not as specific tests for its detection and etiotropic treatment. Vilyui encephalomyelitis is the disease calling the relentless universal scientific interest in it, both in Russia and in the World, as evidenced by numerous publications in Russian and foreign literature. An evidence of this are the words spoken by the professor, laureate of the Nobel Prize in Physiology and Medicine in 1976 Member of the National Academy USA of D. Carleton Gajdusek: «Every neurologist in Europe and the United States knows about Viliuisk encephalomyelitis from our publications in journals «Brain» and «Science», of our lectures, and from the chapter on Vilyui encephalomyelitis of the textbook Richard Johnson on Infectious Diseases of the nervous system. In 2014 a group of authors headed by L.G. Goldfarb (National Institute of Neurological Disorders and Stroke, Bethesda, Maryland, USA) – N.M. Renwick (Rockefeller University, New York), V.A. Vladimirtsev and F.A. Platonov (Institute of Health NEFU), prepared for the publication of a joint monograph "Viliuisk encephalomyelitis", which is dedicated to the memory of Procopius, A. Petrov, who was a pioneer-discoverer of the Viliuisk encephalomyelitis, "the most loyal and consistent knight science", by definition of D.C.

Gajdusek. This monograph summarizes all known data on the study of this disease for more than 60 years, highlighted the pathogenesis of the disease. The release of the monograph is the result of long-term and comprehensive study of this deadly disease. In addition, it will help practitioners - neurologists in the differential diagnosis of a large variety of inflammatory and neurodegenerative diseases of the nervous system, as well as for the medical examination of new cases and patients with chronic disease.

As the result of development of the VEM as the panencephalitis, most patients receive a combination of pyramidal, extrapyramidal and cerebellar disorders, which for a post mortem examination corresponds to lesions in the subcortical gray matter formations: a black substance, own nuclei of the pons, the inferior olive, reticular formation of the pons, medulla and cerebellum. As a result of diffuse lesions of these structures, the disease is accompanied by a wide clinical polymorphism and thus quite often - the development of the syndrome of Amyotrophic Lateral Sclerosis in the acute [16] and the terminal stages of the disease [12,14,15,18]. Syndrome of Amyotrophic Lateral Sclerosis is caused by destruction of the gray matter of the spinal cord in the anterior horn and the intermedial part.

Amyotrophic Lateral Sclerosis (ALS) is a severe neurodegenerative disease, which is accompanied by the electoral defeat of the Central and peripheral motor neurons, rapidly progressive course with the inevitable death. Despite the study of this fatal disease since the late Nineteenth Century, the etiopathogenesis of ALS, as well as VEM, to date, remains poorly understood [6].

Recent observations of the occurrence of cases of VEM and epidemiological studies of ALS, conducted in Yakutia, show a significant decline an epidemic VEM over the last 20-year period on the one hand, and a tendency to increase the incidence of ALS in the Sakha Republic, on the other hand [5]. However, given the significant number of patients with chronic forms of VEM, living in the different republican regions, the increasing migration of the indigenous population, both inside the country and outside it, and the Russian Federation does not exclude the possibility of the spread of the disease around the World. The clinical polymorphism and heterogeneity of VEM in the subacute and chronic phases of the disease, rare new typical cases of the disease at the present time, clinically weakly expressed latent current forms, the absence of specific laboratory diagnostic tests, as in VEM and ALS, can present certain difficulties in the practice of physician-neurologist. The VEM clinical polymorphism is described in the works of P. A. Petrov [15], A.N. Shapoval [20], A.I. Vladimirtsev [8], and others researchers.

A.I. Vladimirtsev included syndrome ALS in one of the 8 chronic forms proposed VEM classification [8]:

1. Dementive-paretic;
2. Spastic-paretic;
3. Syndrome protracted infectious psychosis;
4. Syndrome of amyotrophic lateral sclerosis;
5. Cerebellar
6. Parkinsonoid;
7. Diencephalic;
8. Pseudoneuroasthenic.

P.A. Petrov (1987) divided the chronic form with a slow long over, after acute and subacute forms of VEM on [15]:

1. Chronic panencephalomyelitis;

2. Protracted illness;
3. Amyotrophic Lateral Sclerosis;
In his dissertation P.A. Petrov notes that the syndrome of ALS clinic of VEM, increasing the severity of the disease, leading to rapid progression and irreversible fatal [16].

In our days with the diagnosis of IEM in the first place, you need to follow the diagnostic criteria that have been processed in accordance with the recommendation of the world Health Organization [9] and finalized in [10].

In accordance with the diagnostic criteria can be set definite, probable and possible (doubtful) VEM.

Definite VEM installed in the presence of typical manifestations in patients with the following three forms:
A. The acute form (rapidly progressive), with the course of the disease less than 12 months, which is characterized by:
1. Prolonged fever (7 days or more);
2. Symptoms of viral meningoencephalitis with lymphocytic pleocytosis and steady increase in the concentration of protein in the cerebrospinal fluid;
3. Signs of pyramidal system;
4. Pathological examination: lymphocytic infiltration of the meninges with multiple foci of micronecrosis and perivascular infiltrates in the gray matter of the brain and spinal cord;
B. Subacute form (slowly progressive) over the disease from 1 to 6 years:
1. The clinical picture of a slow progression: progressive dementia, dysarthria, signs of pyramidal and extrapyramidal systems;
2. In clinical and laboratory studies: lymphocytic pleocytosis and increase in the concentration of protein in the cerebrospinal fluid; detection of oligoclonal immunoglobulin of cerebrospinal fluid;
3. MRI \ CT images of the brain: diffuse atrophy of the brain;
4 * Pathological examination: Micronecrotic foci, inflammatory changes in the brain parenchyma, perivascular infiltrates and diffuse loss of neurons;
5. Documentary meningoencephalitis history;
* To validate definite VEM morphological study is desirable, although the presence of the above clinical signs is sufficient.
B. The chronic form, with disease duration of more than 6 years with long periods of stabilization of symptoms:
1. The clinical picture observed cognitive impairments of varying severity, dysarthria, signs of pyramidal and extrapyramidal systems;
2. In clinical and laboratory studies:
* oligoclonal immunoglobulin in spinal fluid;
3. MRI / CT images of the brain: diffuse atrophy of the brain.
4. ** Pathological examination: hardening and sometimes residual inflammatory infiltrates in the meninges, the presence in the brain parenchyma microcysts replacing micronecrotic foci, diffuse brain atrophy, gliosis, neuronal loss.
5. Documentary meningoencephalitis in history;
* Intrathecal production of oligoclonal IgG can be observed during the 3 decades after the onset of the disease. Oligoclonal IgG production is stopped when the inflammatory process "burn out"

at the late phase of the disease with the stabilization of the clinical picture and the development of fibrosis / atrophy of brain structures and minimal residual inflammation.

** To validate definite VEM morphological study, it is desirable, although the presence of the above clinical signs is sufficient.

The diagnosis of "probable VEM" can be set, if the disease has not yet developed to the typical picture of the disease, or the patient is not fully examined.

The diagnosis of "possible (doubtful or hypothetical) VEM" is set if there are serious grounds for suspecting the presence of another disease.

However, the above diagnostic criteria VEM unable to provide all the possible variations and combinations of symptoms. Therefore, in the diagnosis, it is necessary to take into account the available medical records, the various manifestations of the disease and paraclinical data of the study. For example: in all forms to set a definite diagnosis VEM is desirable to have documented evidence of meningoencephalitis, which is in a number of reasons specific to the territory of Yakutia, can not be performed in all cases, because of the remoteness of the district centers of medical districts and obstetric points, so and due to the weather conditions encountered. But the mention of about resent neuroinfections (except the patient) can be collected from his relatives, medical personnel and others witnesses this transfered illness and to make the data in the medical records.

According to the above criteria in a database on 01.01.2014, were included 356 cases with reliable VEM, of which the living are now 110 patients with spastic-paretic, dementive-paretic, and psychotic forms of the disease and 41 patients with various forms of VEM, including the syndrome of Lateral Amyotrophic Sclerosis.

Substantial diagnostic difficulties arising due to the clinical polymorphism of VEM may occur in the chronic phase of the disease. As is known, Viliuisk encephalomyelitis, as Amyotrophic Lateral Sclerosis, may give bulbar and pseudobulbar developing syndromes and muscle atrophy. Speech disorders and swallowing occur in patients with VEM and ALS due to the combined spastic bulbar and pseudobulbar syndromes in lesions of supranuclear and nuclear caudal group of cranial nerves. Patients are also observed dysphonia, drooling, impaired chewing and swallowing, increased facial reflexes, weakness and atrophy of the muscles of the tongue and soft palate, inappropriate laughter, uncontrollable crying loud, etc. Neurodegenerative disruptions of the motor neurons of anterior horns of spinal cord cause of muscle atrophy.

V.A. Vladimirtsev also described damage to the peripheral motor neuron in the global electromyogram (EMG) in the terminal phase of each of the clinical forms of VEM. He developed the technique of parallel conducting global and stimulation EMG allowed us to identify signs of dysfunction of the peripheral spinal neurons in 164 patients VEM, which is used to predict the development of more severe spinal amyotrophic syndromes, including ALS, in some VEM cases [4]. Follow-up studies TY Nikolaeva [11] showed that the syndrome of Amyotrophic Lateral Sclerosis is observed in the later stages of the VEM and more often in the terminal stage, in the context of existing symptoms of the pyramidal and extrapyramidal systems. Clinical similarity of chronic VEM with classic ALS may occur in cases of development without the typical acute onset. Therefore differential diagnosis syndrome ALS of VEM with a gradual disease onset even in a stable epidemic VEM focus is very difficult. Although P.A. Petrov observed the development of the syndrome of ALS, as in the subacute and chronic stages of the disease [16]. In his dissertation, he gives a description of two clinical cases: in the first case, the patient signs of bulbar nuclei appeared after 9 months after undergoing prolonged fever for 10

days and drowsiness. Later joined by symptoms of both central and peripheral motor neuron doing fatal outcome within 25 months from the onset of the disease. Such an onset and course of the disease was observed even in 4 patients. In the second described case, when bulbar syndrome developed in the acute period of the woman during the same long hectic 10-day period as 1 if the patient has developed dysphonia and dysarthria. After stabilization, she went back to work, but after a year the state has gotten progressively worse: bulbar symptoms increased, symptoms of pseudobulbar palsy joined, pyramidal and extrapyramidal symptoms, atrophy and fibrillation in the muscles of the arms and shoulder girdle and intellectual impairment, transformed into dementia. Fatal outcome was 29 months from the onset of the disease. Bulbar syndrome that develops in the acute period of VEM, P.A. Petrov still observed in 3 patients.

Among the patients included in the database VEM, dysarthria observed in 87% of cases, dysphagia in 5% of cases, and muscle atrophy in 17% of cases. As can be seen, slurred speech and muscle atrophy occur quite often. In the differential diagnosis between the VEM, which is accompanied by the syndrome of Amyotrophic Lateral Sclerosis and ALS classic, you need to consider the development of intellectual decline, extrapyramidal and cerebellar symptoms in Viliuisk encephalomyelitis, which is not characteristic of ALS. Also one of the distinguishing features may be unusually long duration for syndrome ALS of VEM sometimes (13 years in one such case for example according to our observations). The main manifestations of chronic phase VEM: intellectual impairment, personality change, often psychopathological conditions, movement disorders in the form of spastic paresis predominantly of the lower limbs, impairment of the function of the cranial nerves, bulbar and pseudobulbar disorders, decreased visual acuity, concentric narrowing of the visual field, expressed endocrine and autonomic disorders sometimes small muscle atrophy.

In the differential diagnosis of Amyotrophic Lateral Sclerosis can also help detect intrathecal production of oligoclonal IgG, which is stably present in the 3 decades after the onset of the disease. [17, 21] This technique is implemented in practice of Yakut neurologists from 2011 in the differential diagnosis between inflammatory and neurodegenerative disease of the brain. Tapping on electrophoregram cerebrospinal fluid of individual bands corresponding to clones of immunoglobulins assessed as positive (Fig. 1 (electrophoretogram)).

Imaging on magnetic resonance and computed tomography of the brain during VEM discover communicating hydrocephalus, increased lateral and third ventricles, diffuse atrophy of the cortex, mainly in the fronto-parietal-temporal areas [8] (Fig. 2 CT images)

Thus, in the differential diagnosis between VEM ALS syndrome and classic ALS, careful history taking to identify acute and subacute inflammatory period, the first appearance of symptoms at the onset of the disease, to assess the epidemiological situation in the place of residence at the time of disease and currently hold tracking the migration of the patient. In identifying patients with suspected or ALS VEM, it needs to carry out a full clinical examination with the use of neuroimaging techniques and definitions oligoclonal IgG in the cerebrospinal fluid.

The authors of this article hope that it will help to find the differential diagnostic criteria in the diagnosis of serious diseases and the development of therapeutic approaches for the optimal management of patients.

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1 2

Fig.1. Electrophoregram IgG of serum and CSF of patients with oligoclonal IgG and IgG without oligoclonal

1. - patients with oligoclonal IgG

2. - patients without oligoclonal IgG

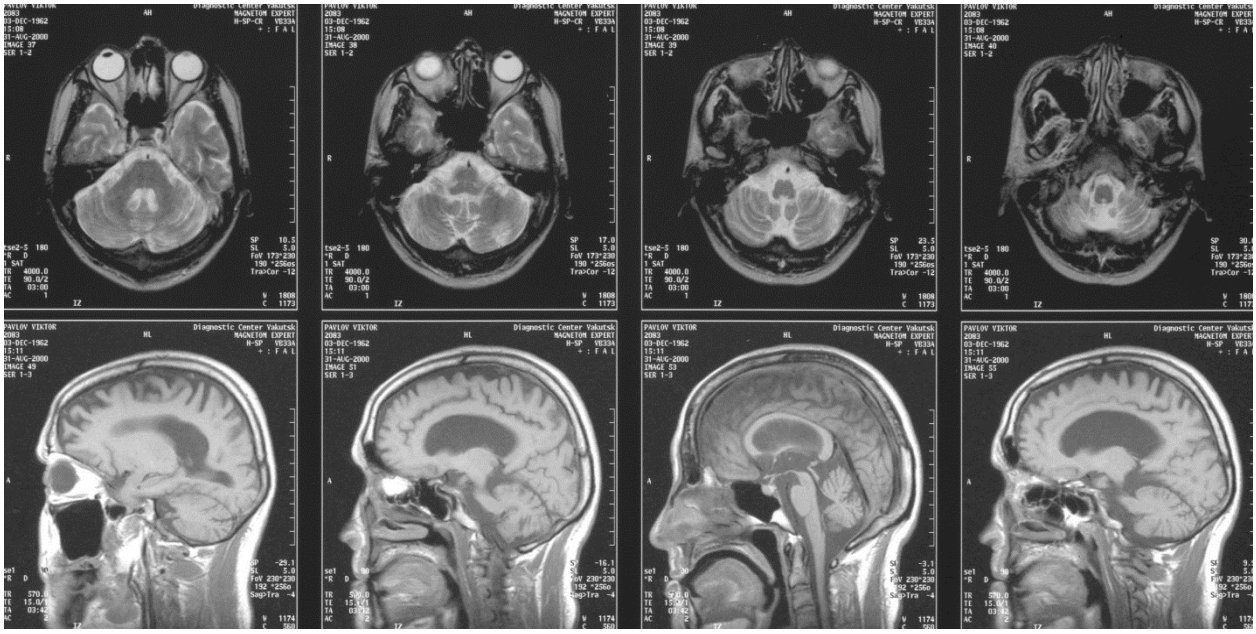

Fig. 2. CT scan of a patient with chronic VEM. Pronounced normal pressure hydrocephalus, cortical atrophy of the brain and cerebellum (V.A. Vladimirtsev 2013)