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Features of Clinical Picture of Viliuisk Encephalomyelitis at the Present Stage

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Resume: The author gives classification of VE and examples of clinical cases of spastic-paretic and demented-paretic forms of VE. At present some changes in severity of VE development in patients are revealed. Lately clinical manifestations of VE have changed a little to ease. Primary chronic forms of VE without acute phase are found out.

Keywords: Viliuisk Encephalomyelitis (VE), spastic-paretic form (SPF), demented-paretic form (DPF).

In the permanent loci of Viliuisk Encephalomyelitis (VE) the contingent of patients is revealed with encephalopathy of unclear genesis, organic neurologic microsymptoms (ONMS) that are typical for clinical authentic VE cases [1]. Such state of health of the group of VE risk

usually found out at prophylactic medical examinations of the population is designated by abbreviation «ONMS». During long study and treatment of VE patients it became clear that gradual formation of the nuclear VE syndrome is possible in a smaller part of VE patients without more authentic acute period of illness. It is rather difficult to carry out complex differential diagnostics by practical neurologists at places and consequently examination of the patients in specialized neurologic clinic is required. When encephalopathy of unclear etiology is observed in a patient except for residual, posttraumatic, dyscircular then the fact of contact with typical VE patient and area of residing are of special significance. In the course of life atherosclerotic changes of vessels of brain or some other age disorders can develop in a person that only complicates diagnostics of typical VE. When the typical syndrome of authentic VE expressed in dysarthria, dementia and spastic tetraparesis is vividly formed in a patient it can appear that the further treatment is ineffectual because of irreversible changes of neurodegenerative phase of VE.

The typical for VE are spastic-paretic and demented-paretic forms. For VE diagnostics we were concerned with such clinical displays as disorders of cerebral nerves, speech frustration, dementia and movement disorders.

Spastic-paretic form of VE clinically shows itself in cerebral nerves lesions (III, IV, VII, XII pairs), decrease of memory, movement disorders (paresis, paralyses of different severity).

Demented-paretic form of VE is clinically manifested in disorders of cerebral nerves (III, IV, VII, XII pairs), bulbar affections, dementia (memory test), movement disorders (paresis, paralyses of different severity).

In 1964 A.I. Vladimirtsev [2] on the basis of clinical examination, observation and treatment of VE patients improved previous classifications of VE where he introduced for the first time such forms of chronic VE as demented-paretic and spastic-paretic and described separate syndromes that are characteristic of the given pathology (Table 1).

In 1985 A.I. Vladimirtsev described for the first time and distinguished between typical and out-patient forms of VE in his dissertation thesis «Clinical-epidemiological Observations in the Loci of Viliuisk Encephalomyelitis».

So called out-patient forms of VE are more often registered in relatives and the nearest environment of VE patients. Diagnostic criterion of the out-patient VE form is neurologic microsymptoms. We certain the symptoms that are characteristic for the out-patient VE form: they are lesions in III, IV, VII, XII pairs of cerebral nerves. As it was already noted the out-patient VE forms are diagnosed in comparative aspect to typical VE forms.

Expressed pathomorphosis of typical VE forms has revealed lately some tendency towards mildness of VE course, the number of acute VE cases decreased, the social adaptation of VE patients has become longer. For illustration of demented-paretic forms of VE the description of clinical VE cases are given below.

Clinical example 1.

The patient G., yakut by nationality, was born in 1943 in the village of Chochu in Viliuisk region where she had been living up to 1953. Since 1959r she constantly lived in the town of Viliuisk. She worked as nursing orderly in children's hospital of Viliuisk. The diagnosis: **Chronic Viliuisk encephalomyelitis, demented-paretic form.** The invalid of II group since 1973. At the age of 5 she had measles, right otitis and in 1951 she suffered trachoma. The beginning of VE was acute in May 1972 (at the age of 29). On the 24-th of May appeared the acute symptoms of respiratory virus infection: headache in temporal and frontal areas, dizziness, cold, cough, nausea and vomiting during a week, general indisposition, expressed general

weakness. During 10 days she could not even get up from her bed. Body temperature was 38-39° within 20 days and then it became subfebrile and remained so till September 1972. She was treated in therapeutic department of the central regional hospital in Viliuisk. She left the hospital in June 1972 but she still had dizziness, somnolence till September 1972 and headache - till 1975 (for 3 years). She was treated during 14 days at the neurological department of Viliuisk hospital. In August 1972 weakness in lower extremities appeared. She was sent to the encephalitic department of the republican hospital in Yakutsk (from 18 November 1972 till 24 February 1973 - 98 days). The neurologic status in 1973: Consciousness was kept. Intellect was not lowered. Memory was lowered. Palpebral fissure was D> S. Movements of eyeballs were free. Convergence was weakened at the left. Anisocoria, eyes pupils were S> D. Mandibular reflex was active. Hearing was not lowered. Dizziness was moderate, changeable. The soft palate was mobile. Uvula was straight. Pharyngeal reflex of the palate was lowered. Tongue was slightly moved to the right, fibrillation of tongue muscles was not present. Taste was kept. Swallowing was normal. The right nasolabial fold was smoothed. Active and passive movements in extremities were full. Contractures were not present. The lower right symptom of Bare was outlined. Gait was spastic, slightly slowed down. In a month dynamics usual gait was marked. In Romberg position was steady. Coordination tests were carried out satisfactorily. Skin abdominal reflexes were absent. Tendon reflexes were increased with wide zones in arms D> S, knee reflexes were high, slightly asymmetrical. Ankle reflexes were high, on the right were more vivid, with clonus of feet. Hoffmann's pathological reflexes, Rossolimo's reflexes were strongly expressed. Feet flexory pathological signs were present, D=S. Muscles tonus was slightly increased according to the mixed type, up to the 1-st degree in legs. Fasciculation was absent. Acute neuroinfection (VE) with cerebral arachnoiditis were differentiated. According to data of PEG from 18 December 1972: **Diffuse atrophy of cerebral hemispheres of brain.** Clinical analysis was carried out and the diagnosis was made: Subacute stage of VE.

In 1975r in neurologic status increase of symptoms was registered: tendon reflexes were more expressed, extensive pathological signs were marked, flexory signs of all the group were increased in feet up to 4+. Gait remained normal.

In 1979 - expressed dysarthria, indistinct speech, rough decrease of memory, euphoria, typical spastic-paretic gait. Tonus in extremities was increased, pyramidal - mixed, strong increase of reflexes with slight asymmetry, clonus of feet. Symptoms of dynamic ataxia were slightly expressed. Pathological extensive and flexory groups were caused in feet.

In 1984r - the clinical symptoms were still increasing: dysarthria, indistinct speech, it was difficult to communicate with the patient. Weakness of convergence was more expressed at the left. The right nasolabial fold was smoothed. There was deviation of tongue to the right. Tonus in extremities was increased up to III degree mixed in legs and up to the I degree in hands. Force was lowered to 3 points in hands and legs.

In 1999 – the state of the patient was stable without increase of the complex of symptoms.

In 2002 - without increase of the complex of symptoms.

Thus, the given clinical example demonstrates at first gradual increase of expressiveness of complex of typical VE syndromes, then stabilization of these symptoms, moderate dementia within the limits of psycho-organic a syndrome, slow increase of dysarthria, absence of bulbar symptoms and also movement disorders without increase of spasticity and rigidity.

Clinical features of spastic-paretic VE forms are shown in the example 2.

Clinical example 2.

The patient S., yakut by nationality, was born in 1950 and lived in the village of Njurbachaan in Njurba region. She worked as a nurse in the kindergarten of Njurbachaan. The diagnosis: **Chronic VE, spastic-paretic form.** The invalid of II group since 1990. She denies the acute onset of disease. In 1975 (when she was 25 years old) there was the episode of her suffering of sleeplessness during 2 months. In 1980 (at the age of 30) weakness in legs appeared and she went to a neurologist in Njurba because of this weakness and was treated there with the diagnosis: lumbar osteochondrosis. In 1987 (at the age of 37) at prophylactic medical examination by a neurologist rough organic neurologic symptoms were revealed and the patient was sent to encephalitic department of Yakutsk republican hospital. She was taken to the encephalitic department for the first time in 1987. Since 1988 was treated annually in specialized VE clinic. Gradual increase of spasticity in legs was observed that allowed in 1990 to prove the diagnosis of chronic VE, spastic-paretic form. MRT of brain in 1998r in comparison to 1995: **CT symptoms of cerebral atrophy of the mixed type.** According to clinical data of the same period there were initial signs of psycho-organic syndrome, increasing dysarthria, lower spastic paraparesis.

The neurologic status in 1998: Palpebral fissure was D> S. Convergence was weakened at the both sides. The right nasolabial fold was smoothed. Pharyngeal reflex was lowered. Speech was dysarthric. Tongue was straight, atrophic at sides. Muscles of shin were asymmetrical (hypotrophy of muscles at the right). There was fasciculation in calf muscles of feet at mechanical irritation. Gait was spastic-paretic. Skin abdominal reflexes were absent. Tendon and periosteal reflexes in arms were D> S with widening of reflexogenic zones. Hoffmann's pathological hand reflexes and Rossolimo's reflexes were expressed. Knee reflexes were high, D> S. Ankle reflexes were strong, at the right side they were higher with clonus at both sides. Feet flexory pathological signs of Rossolimo, Zhukovski, Bekhterev were present at both sides. Reflexes of Babinski, Chaddock were present, D> S. Reflexes of oral automatism were expressed. Tonus of muscles was high in legs according to the mixed type. Hypesthesia in feet and shins was according to socks type.

In 1998-2002 the state of the patient was stable without increase of the complex of symptoms.

In 2005-2008 increase of symptomatic complex, highly expressed spasticity of lower extremities, pathological signs of extensive and flexory group were caused in legs. Tonus in arms and especially in legs was high and of mixed type.

Thus, the clinical example 2 of spastic-paretic form of VE shows the same complex of symptoms as demented-paretic form but without dementia.

Lately clinical features of VE have changed a little. If in 1960s acute and rapid progressive VE forms prevailed then during the last 20 years moderate, slowly progredient forms of VE with long periods of remission are more frequently observed in patients. Primarily chronic forms of VE omitting acute phase of illness have become more frequent at present.

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Table 1

The author	Year	Classification
Vladimirtsev A.I.	1964	Chronic stage, forms: a) demented-paretic b) spastic-paretic c) syndrome of long infectious psychosis d) syndrome BAS e) cerebellar f) parkinsonian-hyperkinetic g) diencephalic h) pseudo-neurotic.

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