CLINICAL CASE

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POSSIBLE CAUSES OF RARE DISEASES IN RELATIVELY SMALL ETHNIC GROUPS. QUESTIONS OF MIXED PATHOLOGY AND DIFFERENTIAL DIAGNOSIS

ABSTRACT

The presented clinical case is probable case of the Creutzfeldt-Jakob Disease (CJD) from the region of distribution of the Vilyui encephalomyelitis (VEM) in Yakutia. The disease occurs in the form of rapidly progressive, fatal meningo-encephalitis, with positive identification of oligo bands of IgG in the cerebrospinal fluid and meningeal symptoms, but with CJD characteristic MRI neuroimage changes. Such cases, even being single and possibly not related to prion diseases, should cause the attention of the scientific and medical community and require thorough investigation and further study.

Keywords: Vilyui encephalomyelitis (VEM); Creutzfeldt-Jakob Disease (CJD); epidemic process; torpid encephalopathy; prion encephalopathy; slow infection; clinical polymorphism.

INTRODUCTION

On the territory of the Republic of Sakha (Yakutia) has particularly high levels of morbidity and mortality from neurodegenerative diseases (1,4,6,12). This group of diseases include: Epilepsy, Parkinson's disease, Multiple Sclerosis (MS), Amyotrophic Lateral Sclerosis (ALS), Spinocerebellar Ataxia (SCA), Myotonic Dystrophy (MD), Charcot-Marie-Toots (SMT), Hereditary Spastic Paraplegia (SP), and other more rare diseases, as Vilyui encephalomyelitis (VEM), which was more widespread for three decades ago (Fig. 1).

Noteworthy that VEM spreading in areas with above-mentioned diseases, had and has, as a rule, the late diagnostics due to clinical similarities of its chronic neurodegenerative stage with a stationary current.

Often in the 1950s-1970s a multi-year duration of a relatively benign stage of such encephalopathy with normotensive hydrocephalus (11) was completed with the accession of af the terrible syndrome of amyotrophic lateral syndrome (ALS), Parkinson Disease syndrome with marked rigidity, spasticity like Strumpell disease.

In some settlements, conspicuous VEM foci, was observed the occurrence of polymorphic

typical and atypical clinical forms of the disease in contact with each other non-family members on occasion for over 10 years. Thus, in the village of S., located near the Vilyuy district (where historically were first reported VEM cases), there was a case of chronic VEM with typical clinical manifestations of cerebral forms of ALS with duration for the 13th years. Such cases still cause a lot of questions, because in most cases the syndrome ALS of the VEM is markedly different from the disease ALS. Despite all the advances in ALS genetics, etiology approximately of the 60% of cases remains unknown. It is assumed that the ALS in the Yakut population is caused by a single genetic variant (7). How does this "pure ALS" case had appeared in the surrounding of the five other confirmed VEM cases in this rural epidemic remains a mystery.

On the basis of numerous discussions in specially created commissions diagnostic VEM criteria were again revised in 1996 and finalized in 2000 year (2). Constant attention to the improvement of diagnostic criteria was important because over the past 60 years the VEM clinical features has changed (tab.1). The acute form of the disease accounted for the majority of patients in the 1950-ies (4,9,10) and in many cases leading to death within several weeks to several months, but over time more and more patients overcome an acute phase. In the 1960s and 1970s, half of newly registered patients developed subacute forms, and in the 1980s and 1990s, almost all registered patients overcome acute and subacute phase and passed in chronic. Long-term clinical-epidemiological observations suggest that simultaneous mosaic morbidity by the practically not registered latent and primary chronic, acute, subacute, secondary chronic forms and slow fatal VEM infection defines special character of an undulipodia epidemic VEM process (1,3). It is difficult to exclude the spread of VEM infection as it is like under the herpes simplex virus (HSV) infection, despite significant clinical, morphological and virological differences from herpetic encephalitis, but with positive oligoclonal IgG antibodies to HSV in CSF from typical VEM patients (8).

Possible research work with microRNA (4,7) will provide the answers to these exciting questions, among which the emergence of multiple sclerosis in the Yakut-Sakha only in the late 1980s. And in this case, is complete without mention of the VEM: at those time, there was registered a case of eventually development of neuromyelitis optica (Devic's Disease) from the young woman who survived after the typical acute VEM, followed 5 years, gradually progressing torpid en-

The distribution of cases VEM according to clinical forms in different periods of registration

Decades	Progressive acute and subacute forms	Chronic forms with acute onset	Chronic form, with a gradual onset	All registered VEM patients
1960–1969	20 (29)	34 (42)	27 (30)	81
1970–1979	58 (49)	31 (26)	30 (25)	119
1980–1989	3 (7)	18 (43)	21 (50)	42

cephalopathy characterized by severe mental depressive disorders and asthenic syndrome. It is possible that a detailed study of the ethnic predisposition to VEM will help to shed light on the effects of population relationships VEM mysterious virus and its host, likely contributing to increased immune vulnerability of the Central nervous system "healthy" carriers and the possibility of severe mixed pathology in these cases.

Clinical observation

In confirmation of the above, it is possible to result a case of rapidly progressive meningoencephalitis in woman 53 years, beginning in November 2013, ended in death on March 3, 2014 with clinical and neuroimaging signs similar to probable Creutzfeldt-Jakob disease, but with positive IgG oligoclonal bands in CSF and meningeal symptoms that denies the possibility CJD.

Patient GGG, date of birth 13.05.1960/53yrs, from the village Tchineke, Vilyuisky district, was admitted to the neurological Department of the Republican hospital No. 2 (the Republican Center of Emergency Medical Care) 10.02.2014, delivered in sanitary aviation, in critical condition with the referral diagnosis "Vilyui encephalomyelitis, tetraparesis".

The disease anamnesis: From the beginning of November 2013. the patient had severe dizziness, disturbances of gait, in the course of the month she was at home, periodically examined by a paramedic, appointed Cavinton, Piracetam. On 26.12.13 she was hospitalized in the neurological bed of therapeutic department of Cenral Region Hospital in Vilyuisk. From the words of older sister of the patient, then she became lethargic, there was dysarthria, marked unsteadiness of gait, weakness in the limbs, walking with support, there was confusion. Contact with the patient was difficult, barely answered questions, no longer recognize her daughter, she developed sleep disorders, pelvic disorders by type of incontinence, stool.

According to her daughter the mother did sick in June-July 2013, she complained of dizziness, were associated with undernutrition. In November 2013, the daughter came home on vacation and noticed that mother became ill to walk – when walking suddenly «dropped» on one or the other leg. While not held in the hand of the subjects, poured the tea. In December 2013, she barely moved. Dragged her feet, was unstable, with difficulty sitting, bad, her speech became incoherent with slowly words pronounciation. Claimed that her long dead mother is at home nearby. When the daughter lived in Yakutsk, she mistakenly told her sister that her daughter is in the next room. December 26, 2014 when admission to CRH were still talking, not moved since January 2014 did not recognize loved ones, was completely immobile, verbal contact was absent, she had also bad swallowing.

In neurological status from 26.12.2013 – pupils uniform, live reaction to light. It doesn't follow the hammer. Hypomimia. The tongue does not show. Reflexes are high with polikinesia. Muscle tone in the limbs high, oral automatic reaflexes are positive. The Babinski symptoms caused easily. No sitting. No active movements observed. No control over urination and defecation. Received Mexidol 4 ml/drip No. 5, Gliatilin 4 ml/No. 5, Vitamin B6 3 ml Dexamethasone 4 mg/m No. 5, Cavinton inside,Prednisolone.

Optometrist CRH consultation – angiopathy of the retina.

Brain CT from 20/01/2014, Conclusion: Foci of pathological density in substance of the brain, brainstem and cerebellum is not revealed.

The history of life: She was Born in the village of Tchinece of Vilyui district, 5th child of 6. The parents: mother died of old age (80 years), father was a party to the World War II, died at 70 years of age, neurological diseases were not sick. Nobody known about VEM and other neurological diseases In their families. One of the brothers of GGG patient for many years suffers from a mental disease with dementia and epileptiform fits and is constantly in a Psychiatric Hospital in Vilyuisk (PND). Neurological examination of the GGG patient's daughter from 26.02.2014 also identified the symptoms of encephalopathy OHMS 3 degrees in the form of a light double sided pyramidal insufficiency, on brain MRI moderate expansion of furrows on the convex are present.

Marital status – not married, has a 22 yrs old daughter.

Graduate librarian (studied in Ulan-Ude). Specialty was not working. Previously worked as a milkmaid (one year after high school), then as a cloakroom attendant in the Verkhne-Vilyuisk in a few months, then returned to his native village, watching the house. Recent years anywhere did not work, lived with the daughter of a native elder sister dependent on her.

The transferred diseases ulcerative colitis (2007).

No injuries there. Tick bite denied. Surgery was not.

Blood transfusion in 1992, about postpartum complications. After that, always felt weak, she complained of headaches.

In the reports of the Vilyui district neurologist of the GGG patient , born in 1960, registered at risk of VEM with organic neurological micro symptoms 3 degrees (ONMS 3) in 1976. Was examined in connection with accommodation next to the patient OTI, 1929 year of birth who had an acute VEM in 1975 and died of disease after 11 months.

Diagnosed with ONMS 3 - meaning the visible pyramidal insufficiency, the patient was on the account at risk of the VEM, according to her daughter, she was frequently bothered by headaches with nausea and vomiting. Since about 2006 there were psycho-emotional disorders, sudden mood swings, aggressiveness.

Objective status: Normal physique. The nose probe is Installed. Skin and visible mucous membranes of normal color, wet. Peripheral lymphoid nods are not enlarged. The breath is held, all fields, weakened in the lower divisions. The heart tones are muffled, rhythmic. AD 110/70 mm hg. The abdomen is soft, painless. Urination in a diaper, a urinary catheter installed. Pastoznost lower limbs. Decubitus ulcer of sacral area is medium in size. Pastoznost both feet.

Neurological status: Paresis of gaze to the left. Pupils D=S, the reactions live. Gaze not followed for the hammer. The face without a clear asymmetry. The tongue does not put out. Caused reflexes of oral automatism. No active movements in the lower extremities. Myoclonus of the hands are seen of from the shoulder joints, increased muscle tone in the limbs, more in the hands S>D. Deep reflexes with hands live D=S, with legs low, D=S. Positive (+) Rossolimo symptom from both foots. Mild symptom Babinski is evoked on the left foot. Coordination tests are failed. Sensitive disorders are not reliably assessed. The viscosity of the neck muscles, Kernig symptoms 80º to the left, 60º right.

Optometrist the Republican Hospital No. 2 dated 11.02.2014 – No contact. The eyelids closed. Paresis of gaze to the left. Epithelial edema of the cornea. Miosis, a sluggish reaction to light. Fundus is not seen by ophthalmoscopy. Diagnosis: Ophthalmoplegia.

Conclusion MRI of the brain from 13.02.2014 – the picture is diffuse hypoxic brain damage, in differential terms

you should be aware of carbon monoxide poisoning, metabolic disorders. Atrophic changes of the cerebellum. Mr-signs of dyscirculatory encephalopathy.

When re-analysis of MRI studies of the brain from 14.02.2014 (fig. 1) identified the following changes: a symmetric increase in the intensity of MR-signal at T2VI, TIRM and diffusion-weighted images (ep_b1000) from shells, heads of the caudate nuclei, pillows and dorsomedial divisions of the thalamus (a symptom of the "hockey stick"). Similar hyperintense MR signal was detected along the parasagittal, just medial cortical parts of the frontal lobes and insular areas. Severe atrophic changes of the cerebellum, with moderate atrophic changes in the Pons, the legs of the brain. Small foci of leucopathy in the white matter of the frontal-parietal lobes on both sides. Ventricular system of the brain is not extended. Backup spaces of the brain is practically not expanded. Relationships in the craniovertebral transition is not broken. On screening MR-angiography circle of Willis: vessels are typical topography and caliber, signs of AVM and aneurysms of cerebral vessels were not identified. After intravenous administration of an onemolar paramagnetic the areas of excessive accumulation of contrast agent in the substance and membranes of the brain were not revealed.

Conclusion MRI studies: given the clinical data obtained and MR-picture of the brain can assume the existence of Creutzfeld-Jakob disease.

EEG from 11.02.2014 – expressed diffuse changes in the EEG without clear focality and asymmetry. EEG monitoring showed flashes of sharp waves on the background slow wave activity.

The patient's condition irreversibly deteriorated. Spasmus oralis was registered as an opercular motion of the chin spazmatics muscles, high mandibular reflex, with clonus of the lower jaw. Myoclonus in both hands, greater in the right, flexor-extensor at the wrist joint and in the elbow, also a weak hyperkinetic movement in his right leg, knee, and ankle joints. Cellular muscle tone in the hands and feet are low, muscles atrophy especially of the calf – the gastrocnemius and peroneal muscles. Pyramid muscle tone during passive movements with "a symptom of a folding knife" in the hands to the 3rd degree (a five-point system), the same in the legs 3rd degre in the left, 4th in the right. For percussion hammer on the crest of the tibia was noted reciprocal bringing the opposite limbs (variant reciprocal pyramidal spinal phenomenon).

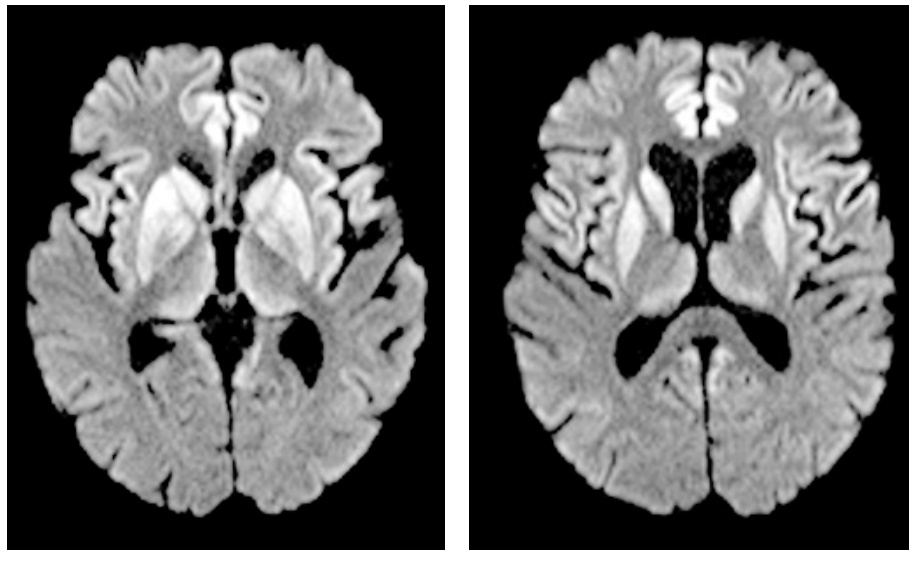

Fig.1-2. MRI photos of patient G., born in 1960. Diffusion-weighted images (DWI) with a coefficient of b1000 determine a symmetric hyperintense signal (restriction of diffusion) from the subcortical nuclei-the heads of the caudate nuclei, the lenticular nuclei (the shell, the pale ball) and the dorsomedial sections of the thalamus (the «hockey stick» symptom). A similar hyperintense signal is noted in the medial cortical areas of the frontal lobes and islet zones on both sides.

Protective reflexes were expressed on the feet more to the right with evoking plantar reflexes. Janiszewski grasping symptom expressed in the left hand. On the feet vividly expressed flexure symptoms of the entire their group. Extensor pathological symptoms were not evoked.

Kernig symptoms on legs right 50º, 80º to the left. Rigidity of muscles of neck to the 3 cross fingers.

The deep reflexes on the arms with bicep evoked live, with expanded area evenly, carpal periosteal evoked when myoclonus were stopped. The knee reflexes has not been evoked. Ankle reflexes low right, left was absent. Without foot clonus.

Abdominal skin reflexes are absent. Red dermographism, wide sharply spreading and resistant. Bedsores on the sacrum, on the skin of the left ankle joint. Central pelvic disorders are expressed.

17.02.2014 analysis of cerebrospinal fluid: With technical difficulties: pinkish, turbid liquor; cytosis= 8/ml^3, protein 16.5mg/dl, sugar was 0.46 mmol/dl, chloride 12.1 mmol/dl, leucocytes sngle in sight, RBC not destroyed 24-28-25 in sight, RBC destroyed -14-18-16 in sight. After centrifugation, the precipitate fell red, transparent.

IgG oligoclonal bands weakly positive in CSF of the patient.

17.02.2014 consultation: Neurological status: She might to respond to the hail, but does not do any job. No oculomotor disorders. Her face without a clear asymmetry. Tetraparesis, to plegia in the legs. Muscle tone is increased along the pyramidal type, more in the legs. Deep reflexes low, D=S. Flexure pathologic foot reflexes (+) . Myoclonus in her hands. No active movements in the legs. She can hold her hands if they are lifting for a while.

The conclusions of the Council: Given the rapidly progressive course of the disease, clinical picture (pronounced pyramidal-cerebellar syndrome, myoclonic seizures, dementia), the typical MRI picture of the disease Creutzfeldt-

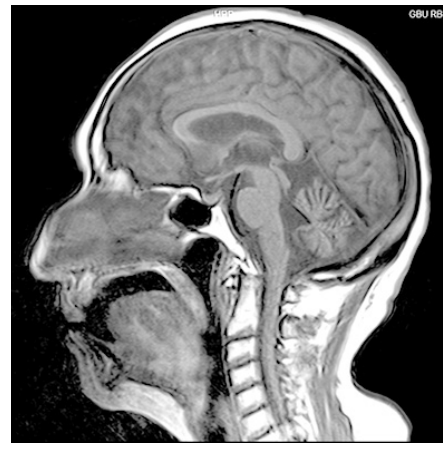

Fig. 3. A picture of MRI of patient G., born in 1960. On the images weighted by T1 in the saggital projection, atrophic changes of the cerebellum, light atrophic changes in the variolium bridge are determined. There is a moderate expansion of basal cisterns. The ratios in the craniovertebral transition are not violated.

Jakob, one can think about likely spongiforme encephalopathy (CJD). Must be differentiated from manifestations of a slow infection of the Vilyui encephalomyelitis.

In the future, the patient's condition continued to deteriorate inexorably. Transferred to the intensive care unit. March 3, 2014, there was a biological death at the phenomena of cardiovascular and respiratory failure.

Discussion and conclusion

A fatal case of the disease the patient GGG in November 2013, March 2014, proceeded on the mixed fast and slow progressive type, with the most pronounced clinical symptoms were not typical for the VEM onset with cerebellar symptoms, very rapidly progressive dementia (globally dementia), myoclonus (very rare when VEM), EEG and brain MRI are typical for CJD. In history marked by the appearance of neurological symptoms from 16 years of age the patient was possible when she could have a contact with neighbor suffering by severe definite acute VEM, which does not exclude a gradual progression of torpid encephalopathy with subsequent exacerbation in 2013. These data, as well as oligoclonal immunoglobulins in the cerebrospinal fluid, meningeal symptoms are not typical for CJD. However, were the precautions taken according to current WHO recommendations when working with CJD patients. Posthumously, with the same precautions held the fence of brain tissue (research in progress).

The disease Creutzfeldt-Jakob refers to prion encephalopathy. The strong similarity of the pathological picture of the brain in prion diseases of humans and in Alzheimer's disease indicates the existence of general mechanisms that lead to changes in neurons and apoptotic death in these incurable diseases. We described amyloid plaques in chronic VEM, which could be senile origin of the 64-year-old patient (3), on the background of the "burned out" inflammatory lesions. A shared mechanism of formation and evolution of amyloid plaques in the brain in Alzheimer's disease and prion diseases, which show involving of neurons, microglial cells and processes of astrocytes. The differences relate to the nature of a protein that is part of the amyloid plaques. In contrast to prion diseases, in Alzheimer's disease there is no evidence of transmissibility. All this suggests that a promising direction of research prion human diseases is a comparative analysis of changes developing in the Central nervous system in other diseases related to neurodegeneration.

Upon detection of morphological features of spongiform encephalopathy in our case (patient GGG), it will be possible to know the mutations of prion protein on the background of torpid encephalopathy VEM as other forms CJD at the moment seem highly questionable. It is not excluded that neurodegenerative stage of the VEM due to persistence unidentified viral agent in predisposing genetically determined, immune defective background of Autonomous immune system of the CNS, in the dynamics of the epidemic process of VEM is favorable background for the development of the first recorded neurodegenerative diseases like multiple sclerosis, leukoencephalitis and even prion encephalopathy. The answer to these intriguing questions is expected in the near future, after virological and morphological studies. However, it is already clear that such cases, even being single and not related to prion diseases, should be of great concern to the scientific and medical community and require careful investigation and further study terrible slow VEM infection. Over the last 3 years in Yakutia found 3 similar cases.

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The authors of this article hope that it will help to find differential diagnostic criteria in the diagnosis of severe rare diseases and to develop therapeutic approaches for the optimal

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CLINICAL EXAMPLE OF A SUCCESSFUL LOCAL THROMBOLYTIC THERAPY AT MASSIVE PULMONARY EMBOLISM

ABSTRACT

This article describes a clinical example of successfully carried out an endovascular local thrombolysis at a massive pulmonary embolism. The carried-out local thrombolysis therapy has surpassed all expected effects of treatment.

Keywords: pulmonary artery, pulmonary embolism, local thrombolytic therapy.

Despite advances in treatment of pulmonary embolism, mortality due to this disease remains very high: 7-8% - of hemodynamically stable patients, 25-33% - of patients with systemic hypotension, 67% or higher - of patients with circulatory collapse, who had pulmonary-cardiac resuscitation [1, 2, 3, 7]. At present, the prevalence of PE is estimated at 0.5 cases per 1000 persons per year. Pulmonary embolism (PE) is one of the most important problems of modern clinical medicine and is the third fatality acute cardiovascular disease [3, 6].

At treatment of PE a priority is elimination of obstruction of a pulmonary artery and restoration of its patency. Hitherto for this purpose used a thrombectomy from a pulmonary artery and thrombolytic therapy [4]. Surgical treatment can save the patient's life with massive obstruction of a pulmonary artery, but really available only to a very small number of specialized vascular clinics [1,4,7]. Currently, the most affordable and the most commonly used method of recanalization of the pulmonary artery in pulmonary embolism, in spite of the obvious flaws, is a method of