A case of rapidly progressive dementia

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INTRODUCTION:
Rapidly progressive dementia (RPD) is a heterogeneous group of diseases, including immune-mediated, infectious and metabolic encephalopathies, as well as prion diseases and atypically rapid manifestations of the more common neurodegenerative diseases. Some of these conditions are treatable and some should be diagnosed immediately due to their potential for infectivity. According to statistics, the occurrence of this disease in the country is infrequent.

Case:
A female patient, 65 years old. Complaints: according to relatives about the lack of speech, the practical absence of any contact with the patient, increased blood pressure, the patient's social maladaptation, the inability to walk independently, the patient's inappropriate behavior. History of the disease: For the first time around January 2022, the patient's relatives noted a sharp decrease in her memory, although she had not previously had such problems. Gradually, a speech disorder, unwillingness to communicate, a sharp decrease in vision joined. The patient was admitted to a specialized neurological clinic. Later she was observed by neurologists with the following diagnoses: dyscirculatory encephalopathy, vascular encephalopathy, astheno-hypochondriac syndrome, chronic cerebral ischemia. For the last 3 months, the patient had a total speech disorder, loss of self-care ability, and behavioral disorders. There is also a history of arterial hypertension with a maximum increase in blood pressure up to 185/100 mm Hg.
Also, in the anamnesis is an increase in anti-TPO up to 1200 IU/ml. She is admitted to the hospital for further examination. The general condition is severe. Consciousness is clear. The position is active. Neurological status: Consciousness is clear. There are no meningeal signs. The pupils are equal in size, photoreactions and corneal reflexes are preserved. There are no oculomotor disorders. The face is asymmetric, with baring slight cerebral palsy VII cranial nerve on the left. Hearing is not reduced. There is no nystagmus. The pharyngeal reflexes are normal. The symptoms of Marinescu-Radovic's oral automatism are positive on both sides. The tongue does not protrude on examination. Muscle strength - difficult to assess objectively due to lack of contact with the patient. Spasmodic hypertonicity in the muscles of the arms and legs. Tendon reflexes are alive, D=S. There are no pathological signs. Sensitivity cannot be tested. Pelvic functions - in a diaper. GNI: elements of mutism, gross cognitive deficit, according to MMSE, MoCA, it is impossible to conduct a study due to total aphasia, agnosia, apraxia. Cerebrospinal fluid analysis: pH: 8, NIT - Nitrites: -, Pro - Protein: trace, Microscopic indicators: *, Leukocytes: Polymorphonuclear leukocytes 0-1 hpf, Red blood cells: 1-2 way, Physical properties: *, Clarity: -, Colour: white, Density: 1.015, Glucose: 100 mg/dl (45-80). In the blood test: thrombocytopenia - 130 * 10 ^ 3 / uL. Conclusion Doppler ultrasound study of the brachiocephalic arteries: Initial atherosclerotic changes in the brachiocephalic arteries. MRI of the brain conclusion: Signs of edema in the projection of the occipital and parietal cortex, posterior sections of the frontal lobes. Diffuse atrophy of the fronto-parietal-temporal region of both hemispheres. Vascular encephalopathy (Fazekas grade II). CT of the brain perfusion results: The cerebral hemispheres are asymmetric. The median structures of the brain are not displaced. The bark and white matter are usually embroidered, their structure is portly. Median structures are not displaced. The convexital subarachnoid spaces of the brain are not dilated. Liquor-containing system: lateral ventricles are asymmetric. The dimensions of the lumen of the ventricles of the brain at the level of the bodies: on the right 1.6 cm, on the left 1.7 cm. The third and fourth ventricles are not deformed. The width of the lumen of the fourth ventricle is 1.1 cm, the third ventricle is 1.0 cm. The Turkish saddle has not been changed. During dynamic perfusion computed tomography: Arteries Right (mm) Left (mm) Anterior cerebral 2.3 2.1 Middle cerebral 2.6 2.6 Posterior cerebral 2.2 2.2 Main artery 3.0 Anterior communicating 1.0 Posterior communicating 1.7 2.0 The lumens of the main arteries are preserved. Sites and zones of pathological accumulation of contrast were not revealed. Conclusion: CT signs of a diffuse decrease in perfusion in both hemispheres of the brain. Pathological changes in the main arteries of the study area were not revealed. EEG: without pathological changes. Diagnosis: Main disease: Neurodegenerative disease. RPD syndrome (rapidly progressive dementia). (F0 3.14) Comorbidities: Hypertensive heart disease III Arterial hypertension II Risk IV. Treatment: 1) Quetiapine 100 mg 1/4 tablet at 9.00 and 1/2 tablet in the evening at 21.00. 2) S. Emoxypine 10 ml + S. Natrii chloridi 0.9%-200 ml intravenous drip once a day at 10.00 daily.3)S. Galantamine 2.5 mg - 1 ml * 2 (2 ml) intramuscularly 1 time per day at 11.00. 4)S. Cholini alfosceras 1000 mg - 4 ml + Sodium chloride 0.9% -100 ml intravenously drip 1 time per day at 15.00. 5) Donepezil 5 mg once a day at 22.00. 5) Magnesium hydroxide + acetylsalicylic acid 75 mg in the evening. 6) Memantine 10 mg 1 tablet at 8.00 and 13.00 7) Bisoprolol 2.5 mg in the evening. 8) Perindopril 8 mg + Indapamide 2.5 mg 1/2 tablet once a day. The dynamics of the patient's consition: a slight improvement - the background of the mood somewhat leveled off, the manifestations of the suffering grimace were noticeably less, the patient reacted better to others. Speech contact with the patient is still impossible.

Discussion:
Diagnosis can be difficult due to its rarity, presentation with nonspecific neurological symptoms, the wide variation associated with them, and the need for extensive examination. Awareness of disease-specific biomarkers, radiological features, and diagnostic criteria is critical for timely diagnosis.

Conclusion:
Unfortunately, there are no effective full treatment options for rapidly progressive dementia, and it is universally fatal, with an average life expectancy of six months. Once the diagnosis is confirmed, clinicians should provide symptomatic treatment for neuropsychiatric symptoms, communicate with the family about the expected adverse outcome, and conduct end-of-life conversations.