MULTIPLE SCLEROSIS. POSSIBLE OPTIONS FOR THE ONSET OF THE DISEASE IN A CLINICAL EXAMPLE

Markovska Olena
Candidate of Medical Sciences, Assistant at the Department of Neurology № 2
Kharkiv National Medical University
Tovazhnyanska Olena
Doctor of Medical Sciences, Full Professor,
Head of the Department of Neurology № 2
Kharkiv National Medical University
Lapshyna Iryna
Candidate of Medical Sciences, Assistant at the Department of Neurology № 2
Kharkiv National Medical University
Nadozirna Yevheniia
Neurologist, communal non-profit enterprise of the Kharkiv Regional Council "
Regional Clinical Hospital"

Abstract. Multiple sclerosis (MS) is a neurological problem of great socioeconomic importance, as it develops mainly in young and middle-aged people and, in the absence of timely and adequate treatment, leads to the formation of severe neurological deficit and early disability of patients in the first year after diagnosis. The clinical manifestations of MS are diverse and the well-known slow onset and development of the disease are not always found. A variety of clinical forms and options for the onset of MS, an increase in the age interval for debut, various rates of disease progression indicate different mechanisms for the development of the demyelinating process and thereby increase interest in studying the clinical features and pathogenetic mechanisms of the development of this pathology.

Keywords: multiple sclerosis, demyelination, atypical debut of the disease.

Multiple sclerosis (MS) is a neurological problem of great socioeconomic importance, as it develops mainly in young and middle-aged people and, in the absence of timely and adequate treatment, leads to the formation of severe neurological deficiency and early disability (up to 80%) of patients already first year after diagnosis [1, 3, 5, 10].

Today, it is generally accepted that MS is a multifactorial disease, in the initiation and development of which an important role is played by a viral infection, a hereditary predisposition, geoecological factors, nutritional features, injuries, and the presence of frequent stressful situations [1, 3]. Numerous experiments and clinical studies have proved that autoimmune disorders are the basis of MS pathogenesis [3, 5, 6]. However, a variety of clinical and morphological manifestations of MS indicate a heterogeneous development of the demyelinating process.

The latter is confirmed by pathomorphological and histochemical studies of foci of demyelination and unchanged white matter, which identified several types of pathological changes in MS, which differ in the severity of demyelination, the nature of damage to oligodendrocytes and progenitor cells, the presence of reparative processes in the foci, which suggests various mechanisms for the development of these disorders [9].

A variety of clinical forms and types of MS can also be due to the heterogeneity of myelin [7, 8], since oligodendrocytes are able to form different types of myelin in different parts of the central nervous system, which differ in immunochemical and physicochemical properties. There is an opinion [2, 4] that in MS there is an initial immaturity of myelin, which makes it especially vulnerable to various inflammatory mediators and active forms of oxygen. It is possible that in some cases a violation of the physicochemical properties of myelin precedes the development of immunopathological reactions [11].

Such complexity and versatility of the pathogenesis of MS determines the significant variability of the clinical picture, the variety of typical clinical syndromes and atypical debuts of the disease.

In this work, as a clinical example, we present the medical history of a patient in whom the debut of the disease was characterized by a stroke-like onset.

Extract from the medical history № 12586. Patient M., born in 1965, in 2019 was admitted to the municipal non-profit enterprise of the Kharkiv Regional Council “Regional Clinical Hospital” with a diagnosis of persistent residual symptoms of a stem stroke (2012) with right-sided hemiparesis and vestibulo-atactic syndrome. On admission, he complained of problems in walking, stiffness of movements in the lower extremities, weakness and awkwardness of movements in the upper and lower extremities, more on the right, swelling in the legs, expressed in the evening, difficulty speaking, hearing loss, more on the left, decreased vision. From the medical history it was found: in 2012, after a nervous overstrain against the background of an increase in blood pressure to

220/120 mm Hg, he suddenly felt weakness in his right limbs and difficulty speaking. The patient was admitted to a hospital with a diagnosis of a stem stroke with right-sided hemiparesis and severe vestibulo-atactic syndrome. Against the background of vascular, nootropic and restorative therapy, positive dynamics were noted. In 2013, the patient's condition worsened again. There was a stiffness of movements in the lower extremities, weakness and awkwardness of movements in the upper and lower extremities, more to the right, difficulty speaking. The patient was hospitalized with a diagnosis of repeated acute cerebrovascular accident. Against the background of the therapy, the patient's condition improved. At these stages of treatment and examination of the patient, a CT scan of the brain was performed, which did not reveal pathological changes and foci in the substance of the brain, including foci of a vascular nature. In the winter of 2019, the condition worsened sharply. There was a pronounced stiffness of movements in the lower extremities, difficulty speaking, pronounced weakness and awkwardness of movements in the right limbs. The patient was hospitalized in the municipal non-profit enterprise of the Kharkiv Regional Council "Regional Clinical Hospital". On admission: the general condition of the patient is satisfactory, cardiac activity is rhythmic, heart sounds are muffled, blood pressure is 140/100 mm Hg, vesicular breathing in the lungs, the abdomen is soft, painless on palpation. In neurological status: palpebral fissures D>S, pupils D=S, eyeball movements are somewhat limited in volume. Light asymmetry of the right lower facial muscles, the tongue along the midline. Speech is chanted. Tendon reflexes from the arms - D>S are revitalized, from the legs - D=S are high, abdominal reflexes are absent. Clonuses of the feet. Muscle strength in the right hand - 3.5 points, in the left hand - 4 points, in the lower extremities - 2.5 points. Muscle tone is increased in spastic type. Pathological reflexes of Babinsky, Strumpel from two sides. Coordination tests are performed with a miss. The Romberg test was not carried out due to the inability to stand. There are no sensitive problems. There are no meningeal signs.

Analysis of general clinical studies (general analysis of blood, urine, blood glucose, protein fractions, acute phase indicators) did not reveal significant deviations from the norm. ECG: single ventricular extrasystoles. Mild changes in the myocardium of the left ventricle. Inspection of the therapist: stage 3 hypertension. Left ventricular myocardial hypertrophy. CHD: atherosclerotic cardiosclerosis. Ventricular extrasystolic arrhythmia. CH stage I. Eye examination: retinal angiopathy in both eyes. Amblyopia of the left eye.

The patient underwent an MRI scan: in the white matter of the brain periventricularly, small foci with a low-intensity increase in MR are detected along the corpus collosum with a low-intensity increase in MR - signal in T2-vi. The ventricular system is moderately expanded. The outer subarachnoid spaces are convex and basally somewhat enlarged. Conclusion: signs of a demyelinating process. Moderately pronounced internal hydrocephalus.

An immunological blood test revealed a slight increase in CEC and an increase in the amount of CD 4, in relation to CD 8.

Given the clinical picture of the disease, additional research methods, the diagnosis of persistent residual effects of a post-stroke (2012) was doubtful. Thus, the final diagnosis was established: multiple sclerosis, secondary progressive type of course, with tetraparesis, gross violations of the walking, cerebellar-atactic and bulbar syndromes. EDSS=7.0 points.

In the commentary on this case, we would like to draw attention to the presence of a relatively late onset disease and stroke-like debut that is not characteristic of MS.

Thus, the clinical manifestations of MS are diverse and the well-known slow onset and development of the disease are not always found.

Therefore, the variety of clinical forms and variants of the onset of MS, an increase in the age interval for debut, various rates of disease progression indicate different mechanisms for the development of the demyelinating process and thereby increase interest in studying the clinical features and pathogenetic mechanisms of the development of this pathology.

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